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The Journal of Periodontology
is dedicated to the memory of
Dr. Gillette Hayden
for her pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

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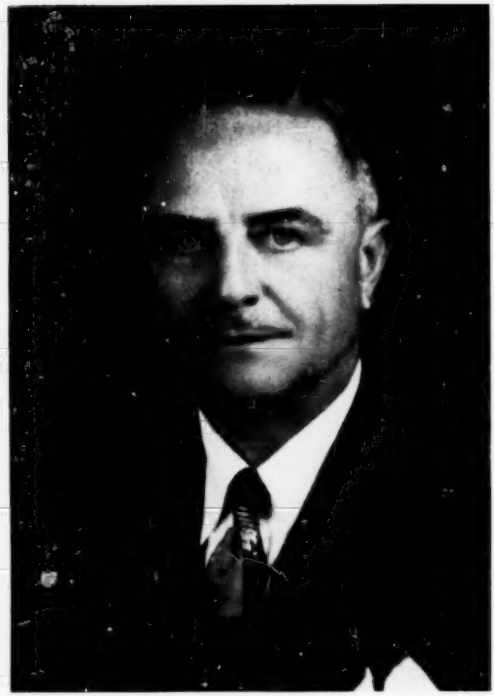


Dickson G. Bell, D.D.S.

President of the American Academy of Periodontology,
1949-1950

Dr. Dickson G. Bell of San Francisco, California, was elected President of the American Academy of Periodontology at its Thirty-third Annual Meeting in San Francisco in October, 1949. Election to this office is the climax to an active career in the American Academy of Periodontology and as a specialist in the practice of Periodontia. That Dr. Dickson merits this honor can be proved by a recitation of his activities: he has specialized in the practice of Periodontia for the past 24 years and is a Diplomate of the American Board of Periodontology; he is a member of the Periodontia staff of the College of Dentistry of the University of California from which he was graduated in 1914; he has been a member of the American Board of Periodontia since its organization; he has been very active in organized dentistry serving one year as chairman of the Periodontia section of the American Dental Association; fellow of the American College of Dentists, member of Omicron Kappa Upsilon and Xi Psi Phi fraternities. Dr. Bell has served on many committees of the American Academy of Periodontology, has presented many papers and clinics before various dental societies, has contributed much to dental literature, and has always been a militant worker for the advancement of the science of Periodontology.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY

The Periodontal Lesion*

The Development of the Lesion and the Establishment and Treatment of the Periodontal Pocket

JAMES NUCKOLLS[1] AND BENJAMIN DIENSTEIN[2] IN COLLABORATION WITH DICKSON G. BELL[3] AND ROBERT W. RULE, JR.[4]

INTRODUCTION

THE PURPOSE of this paper is to present periodontal pathology on the basis of tissue reaction to injury and to emphasize specifically the basic biochemical processes which bring about the disease, rather than the ultimate physical alteration itself. The following presentations should not be considered original concepts per se; rather, they include an integration of thoughts and research derived from many sources. They are essentially some of the many considerations necessary for an understanding of the processes which obtain in periodontal disease.

A sincere effort has been made to accredit those whose works have been utilized. Periodontia must heed the growing application of biochemistry to etiology and treatment, for through the understanding of basic cellular responses to metabolic variations a rational approach to the pathogenesis and therapy of the disease process can be formulated.

The problems concerning the etiology and pathogenesis of periodontal disease have attracted the attention of a multitude of investigators in both clinical and laboratory fields. In general, several concepts have been advanced, and they may be divided into three distinct groupings. The theory which probably has the greatest number of adherents considers that the cause of the disease can be attributed to local conditions in the oral cavity, such as the accumulation of calculus about the teeth, traumatic injury by overstress, open contacts between the teeth, poorly contoured restorations and other conditions which would result in an injury to the tissues of the periodontium.

A less widely accepted theory contends that local disorders are in a great

*Presented at the 7th Pacific Coast Dental Conference, Salt Lake City, Utah, July 13, 1948.

From the University of California College of Dentistry, Section of Oral Pathology and Periodontia.

1. James Nuckolls, Professor of Operative Dentistry, 2. Benjamin Dienstein, Instructor in Operative Dentistry, 3. Dickson G. Bell, Clinical Instructor in Operative Dentistry, 4. Robert W. Rule, Jr., Associate Clinical Professor of Operative Dentistry, University of California Collge of Dentistry, San Francisco.

measure but the reflection of general disorders. A third theory is principally a combination of the two already mentioned, namely, that the disease is a manifestation of both local and systemic disorders. The supporters of these theories are also divided as to methods of treatment. As a result of these controversies, the periodontists have been more concerned with systems of treatment rather than with basic considerations which have to do with disease processes and tissue repair. For example, marked and even revolutionary advances have recently been made in the fields of enzyme chemistry, intermediary and mineral metabolism, and especially in the biochemistry of inflammation. It is not important that every periodontist have a detailed knowledge of all the biochemical changes in disease, but it is most important to have a working concept of these processes—to be able to discuss the condition not only on the basis of macroscopic and microscopic physical changes, but, more important, in terms of biochemical processes that are the basic cause of the physical alteration. With a foundation of these fundamental approaches, it becomes possible to discuss diagnosis and therapy of periodontal disease on a rational basis.

The basis for treatment lies in the endeavor to both remove the causative factors and assist the physiological mechanism of repair and only a thorough understanding of what is actually happening can supply the reasons for preferring one treatment over another.

This paper approaches the problem of periodontal disease from the incipient stage of a disturbed local metabolism brought about by a periodontium subjected to local injury. A distinct demarcation between local and systemic mechanisms is emphasized for the purpose of clarity; however, it is recognized that in many cases where the periodontium is undergoing degenerative changes, the local lesion is complicated by systemic factors, or vice versa. It should be borne in mind that whether the lesion is the result of local factors or caused by a systemic dysfunction, the reaction of the cell is essentially the same. The cell is either normal, slightly sick or completely sick, and it functions accordingly. There is, however, a difference. When the injury is of local nature, only the structures in the immediate area are affected; whereas, in general disorders the metabolism of every cell may be directly or indirectly disturbed to varying degrees.

The teeth and their investing tissues offer one of the most unusual opportunities to observe the reactions of not only epithelium and connective tissue, but of bone, when normal metabolism has been disrupted. An irritant coming in contact with an otherwise normal gingiva will induce an inflammatory reaction which may vary from a barely perceptible hyperemia to an intense suppurative process. Depending on the severity and length of the reaction, the deeper structures of the periodontium may be involved, and there may be a demineralization of the supporting bone. When bone is involved, it is evidenced in the roentgenograph as a marginal rarefaction. If the injury is persistent, there may be a complete breakdown of superficial structures with a concomitant demineralization of bone and loss of the bone matrix. Suppuration, then, is the end result.

THE ANATOMICAL STRUCTURES OF THE PERIODONTIUM

In order to establish a basis for a discussion of the periodontal lesion, a brief review of certain fundamental tissues of the periodontium and their relationships to one another is necessary. Since the anatomical relationship of the root to the socket and the attachment of the periodontal membrane is so well known, no discussion of this will be necessary. Until comparatively recent researches, the precise relationship of the blood supply to the periodontal membrane and the connective tissue overlying the alveolar bone was not sufficiently considered.[1] It has been shown that the main artery supplying the pulp of a tooth divides into two parts—the dental artery and the interalveolar artery. The dental artery is the smaller and supplies the pulp. The larger interalveolar artery courses through the marrow spaces of the

supporting bone and emerges at the crest of the alveoral process as gingival perforating arteries. The gingival perforating arteries are small capillaries which can be followed into the connective tissue papillae of the mucous membrane of the gingiva. As the interalveolar arteries pass through the bone, they give off small branches, the alveolar perforating arteries, which pass through the lateral wall of the alveolar bone proper and supply the periodontal membrane. Each capillary is surrounded by a network of lymph vessels which are the physiological pathway of drainage from the tissues. Both the arterioles and lymphatics play an important role in the inflammatory reaction.

The oral mucous membrane is composed of two layers: the surface epithelium and the lamina propria.[2] The mucous membrane of the gingivae is attached to the periosteum of the alveolar bone by a very dense connective tissue consisting of coarse collagenous bundles. The lamina propria is the dense connective tissue just below the surface epithelium. Its papillae extend outward forming an undulating junction with the epithelium. These papillae contain blood vessels and nerves, some of the latter passing into the epithelium.

The epithelium of the mucous membrane consists of several layers of cells which are derived from the basal layer. The cells undergo a histochemical and morphological change from the basal layer toward the surface. Thus the epithelial structure is maintained by mitotic division of the cells of the basal layer. Under certain conditions, such as irritation, mitosis is observed just above this basal layer in the prickle-cell layer. The granular layer and the keratinous layer complete the transition process. The epithelium of the gingiva is a modified, stratified squamous type. It terminates on the surface of the tooth at the gingival sulcus as the epithelial attachment.

The alveolar process consists of a cortical plate and an interstitial or trabeculated bone. The cortical plate forms the socket and the vestibular and oral plates. That part of the cortical bone which forms the socket is known as the alveolar bone proper and affords attachment to the principal fibers of the periodontal membrane. The trabeculated bone occupies the space between the cortical plates and forms the main body of the process.

THE PERIODONTAL MEMBRANE

The periodontal membrane forms the connective tissue attachment between the cementum of the tooth root and the cortical plate of the bony alveolus; it is continuous with the connective tissue of the gingiva.[2] The tissue elements of the membrane are primarily the principal fibers, fibroblastic cells, the blood vessels, lymphatic and nerves. Epithelial remnants are also present in the membrane, and these are considered to be a part of the formative organ of the root known as Hertwig's epithelial root sheath.

The fibers of the periodontal membrane are composed of white collagenous connective tissue, arranged in definitive groupings: gingival, transseptal and alveolar. Limited mobility of the tooth is allowed by their arrangement which follows a wavy course from bone to cementum. The gingival and transseptal groups are the ones with which the periodontist is most often concerned, since they are the first to undergo degradation in development of the local periodontal lesion.[3] The gingival group passes outward from the cementum and breaks up into small bundles, the fibers of which interlace with the fibrous tissue of the gingiva. The transseptal group extends between the cementum of the roots of the adjacent teeth where the fiber bundles pass over the crest of the alveolus.

THE INFLAMMATORY REACTION*

The inflammatory reaction is the most important of all tissue responses with which the periodontist has to deal. It is the local pivotal reaction around which periodontal lesions have their inception. The subject of

*The study of inflammation is based primarily on the publications of Valy Menkin, and we wish to give full recognition to the author in this regard.

inflammation has always played a dominant role in the history of medicine, but only a few names of those who have contributed to an understanding of inflammation will be mentioned in this paper. John Hunter (1728-1793) advanced two important principles. He believed that inflammation could be caused by any injury, and regarded inflammation as a reparative mechanism which ultimately restored an injured part to normal function. Cohnheim (1839-1884) considered the central point in the inflammatory reaction to be an increased permeability of the capillary wall. Another theory explaining the inflammatory reaction was advanced by Metchnikoff in the early part of the nineteenth century. To him the phagocytic action of leukocytes which disposed of the bacterial irritant was the primary factor in inflammation. The increased permeability of the capillary walls and the migration of leukocytes were considered merely as phenomena of secondary importance. Recent investigations have shown that the theories already mentioned need not be considered separately but, rather, as component parts of a major tissue reaction which is initiated by an impaired cell physiology. This altered cell physiology is in a large measure responsible for the macroscopic and microscopic physical changes.

Since it is now generally recognized that the basic cause of physical alteration is a biochemical process,[4,5] the following discussion will deal largely with this phase.

Inflammation is the manifestation of cellular injury by an irritant. This injury results in a complex vascular, lymphatic and tissue response. An irritant can be any agent that interrupts normal cell metabolism and may be physical in nature, such as calculus, chemical, as the byproducts of microorganisms, or the toxic products of protein breakdown. The inflammatory process consists of many interdependent sequences leading to a localization of the irritant and its ultimate disposal.

The initial response to an irritant is an alteration in the fluid exchange between the capillaries and the intercellular fluid of the immediately surrounding tissue. This is brought about by a momentary capillary spasm followed by vasodilatation and an increase in capillary pressure. The pain of hyperemia and inflammation is probably caused by pressure on sensory nerves in the localized area of vasodilatation.[6] If the cells are injured by the irritant, this reaction is followed by a definite change in the structure of the endothelial wall, increasing its permeability so as to produce a greater fluid infiltration into the tissue. The reaction which causes the increased capillary permeability comes about through the liberation of a chemical unit from the injured cells, called "H substance" by Lewis[7] and "Leukotaxine" by Menkin.[8] This chemical agent can also produce an increase in the permeability of cells in general. The exudate is first serous in nature, but becomes of high protein content when the capillary walls are seriously damaged. A slowing of the blood stream in the affected area accompanies this reaction. In effect, the increase in capillary pressure and permeability produce a seepage of plasma proteins from the circulation into the injured area, in this way elevating the level of the proteins in the exudate. The excessive amount of protein in the area is eventually modified by the rapid rate of local protein breakdown as the inflammatory process progresses. The significance of this will be discussed more fully in subsequent paragraphs.

As the clotting mechanism comes into play, fibrin is liberated from the fluid exudate in the inflammatory area and the local circulation becomes markedly impaired.[9] The small vascular channels are the first to become occluded with fibrin. The lymphatic channels continue to serve as pathways for the removal of colloidal particulate substances from the extra-capillary areas via the lymph nodes and main lymphatic trunks back to the circulating blood. If the inflammation is progressive, the lymphatics become blocked with fibrin a little later, and there is an accumulation of exudate in the tissue resulting in edema. In minor injuries the late

occlusion of the lymphatics accounts for the delayed manifestation of edema. In more severe injuries the rapid occlusion of the lymphatics accounts for the early manifestation of edema. In the more severe injury the occlusion of all vessels as well as the extracellular fluid by the fibrin clot is almost instantaneous and this plays an important role in the immediate walling-off of the injured area. By this means the inflammatory area is localized to an area of varying extent surrounding the injured site.

As a result of the fibrin blockade, or walling-off process, the normal oxygen supply via the circulating blood is cut off and, in turn, the normal oxidation-reduction reactions within the cells are prevented from going towards completion. The injured area then develops its own hydrogen ion concentration and its own metabolism.* As a result of the irritant and the concomitant inflammatory reaction, the metabolic rate increases in the localized area resulting in a rapid depletion of available oxygen and a state of anoxia develops. The want of oxygen in this localized area disturbs the metabolism of carbohydrate which causes the production of lactic acid.[10, 11, 12] The cellular picture apparently is associated with this biochemical change. There is an initial infiltration of polymorphonuclear leukocytes. As the area increases in acidity, the cellular picture changes and macrophages predominate.

In order to develop an understanding of the sequence of events which follows the fibrin blockade, it is necessary to briefly review some of the fundamental aspects of carbohydrate metabolism which are intimately related to the inflammatory reaction (cf. Chart I*).

Glucose, upon entering the tissue cell from the blood stream, "takes up" phosphate and then passes through a series of complex chemical reactions and may be stored in the cell as glycogen, or it may be directly utilized in the production of energy. When the cell is called upon to release energy, the available glycogen or glucose phosphate undergoes a series of extremely rapid reactions to form pyruvic acid. There are two possible pathways by which this reaction can take place. They are known as the aerobic and the anaerobic pathways. The anaerobic pathway is the one which has been studied the more thoroughly and is generally accepted by most chemists.

Pyruvic acid is the principal chemical unit from which energy is derived, but it may be formed from sources other than glucose. For example, it may come from the oxidation of fat when the carbohydrate source is not readily available, or from the various amino acids of proteins (transsamination) (Chart 2). However, glucose is the normal source of supply. Pyruvic acid is broken down in a cycle of chemical reactions known as Kreb's cycle (Chart I). There are many chemical reactions in Kreb's cycle, and in several of these reactions carbon dioxide and two hydrogen ions are split off from the original compound, and a new compound is formed. Thus, there are two principal reactions in Kreb's cycle. One reaction is the removal of hydrogen from the various breakdown products. These hydrogen ions ultimately unite with oxygen from the circulation via another system, the cytochrome system. The end product of this system is the water of metabolism. Energy is released during the transfer of electrons in the cytochrome system (Chart I). The other reaction has to do with the removal of carbon and oxygen from the breakdown product as carbon dioxide. The carbon dioxide is the CO_2 of respiration (Chart I).

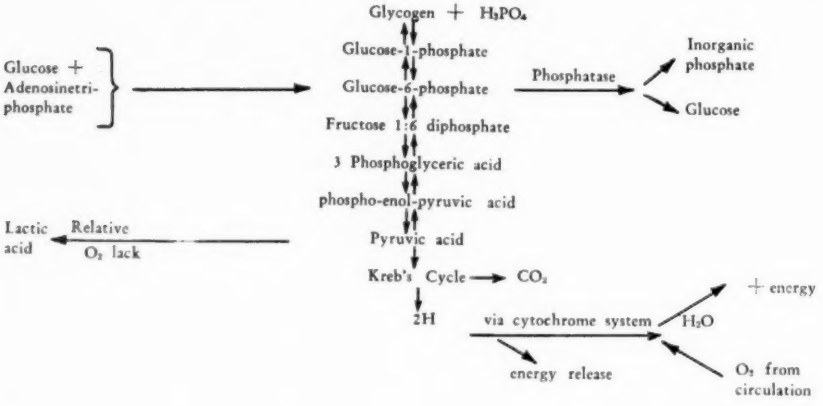
It is recognized that this interjection of a discussion of carbohydrate metabolism is a most cursory one, but it was intended to present only certain fundamentals which must necessarily be referred to in the continued discussion of the inflammatory reaction.

As was previously mentioned, the presence of a fibrin network and thrombosed capillaries and lymphatics not only cuts down the oxygen required by the cells in the inflamed area, but the normal nutrient

*For references to Chart I on Metabolic Pathways and The Cytochrome System see listing in References to Literature.

*CONDENSED OUTLINE OF METABOLIC PATHWAYS**

CHART I



source of glucose is impaired as well. The glycogen reserve in the cells is consequently exhausted, and the energy unit (pyruvic acid) must now be supplied from other sources. This energy requirement for the functioning cells is derived from the breakdown of protein of the plasma exudate. The breakdown and utilization of protein by the cell is known as gluconeogenesis.[13, 14] When the normal oxygen supply is reduced, due to the fixation mechanism, the physiological oxidative process of Kreb's cycle is inhibited, and instead of the normal end products of carbon dioxide and water, lactic acid is formed from pyruvic acid. The CO_2 content of the area of inflammation tends to fall because of the impairment of Kreb's cycle, and this is accompanied by a concomitant drop in the tissue alkali reserve in the area.

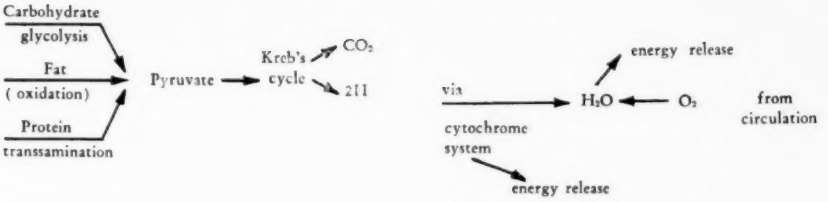
As cellular function continues, there is an increasingly greater accumulation of lactic acid which is not removed by normal channels, and an uncompensated local acidosis develops. With a pH lowered to approximately 7.0 the polymorphonuclear leukocytes become fewer in number and appear as degenerated, swollen and distorted cells. The bulk of the cellular elements is now composed of mononuclear leukocytes. These appear to be fairly normal at a pH of 6.9 to 6.8. If the inflammatory process is unmitigated and the pH falls below 6.5, all types of cells except the lymphocytes are injured and frank pus develops.[5]

From the foregoing discussion, it seems that the suppuration is in part due to the development of a local acidosis. The local acidosis is a result of an impaired glycolysis, gluconeogenesis and a local tissue anoxia. The development of an acid pH activates certain proteolytic enzymes already present in the area, bringing about the ultimate solution of the tissue.

If removal of the irritant and solution of

*For a more complete outline refer to Dienstein and Nuckolls "Metabolic Pathways" from Nuckolls, J. and Rule, R. W., Jr.: Collected Lectures. A Study of the Fundamental Pathology of Periodontal Disease. The Lithotype Process Co., San Francisco, 1947, 1st Ed., p. 15a and 15b.

CHART II



the fibrin occlusion in the vessels is effected, new blood vessels begin to make their appearance along the sides of the wound and form the granulations which are characteristic of tissue repair. An outline of tissue reaction in inflammation is as follows:

Outline of Tissue Reaction in Inflammation
Schematic Outline of Cellular Sequence and Biochemical Factors in Inflammation

1. Irritant.
2. Tissue Injury + reflex + vasoconstriction and transitory increase in capillary pressure followed by a vasodilation.
3. Liberation of Leukotaxine from the injured cells.
4. Increased vessel and cell permeability due to action of leukotaxine.
5. Increased flow of plasma proteins and migration of polymorphonuclear leukocytes—due to increased tissues permeability (tissue pH normal 7.4).
6. Blockage of arterioles by fibrin liberated from the plasma protein.
7. Beginning of edema resulting from increased plasma flow and blocking of lymphatics.
8. Secondarily blockage of extracellular matrix by fibrin and establishment of the fibrin wall.
9. Anoxia due to impaired local lymphatic and vascular circulation + increased cell glycolysis resulting from stimulation caused by tissue injury.
10. Loss of CO_2 content and exhaustion of alkaline reserve beginning gluconeogenesis of plasma protein to form sugar.
11. Incomplete glycolysis and oxygen want due to impaired blood supply with formation of lactic acid instead of CO_2 and H_2O. Drop in pH to 7.0. Change in cellular picture from polymorphonuclear leukocytes to mononuclear leukocytes.
12. Gradual diminution of sugar in exudate as glyconeogenesis continues with increased production of lactic acid and continued lowering of pH to 6.5 when all types of leukocytes degenerate.
13. Liquefaction of tissues from enzymes derived from cells or tissue operating in an acid medium (pus forming).
14. Solution of fibrin blockade and beginning of formation of granulation tissue provided the irritant is removed.

The biochemical changes in bone injury are essentially the same as those of the inflammatory reaction in connective tissue.[15, 16, 17] If this concept is to be accepted as a basis for discussion, we must consider that in the presence of an irritant, the reaction of bone is similar to that of any tissue of mesoblastic origin. The difference between bone and other mesoblastic tissues lies in the former's potential to aggregate mineral salts.[18, 19]

The reaction whereby bone salts make their appearance in the intercellular spaces of a bone matrix is brought about by an enzyme, alkaline phosphatase. This enzyme is present in all tissues undergoing mineralization. It acts on the substrate glucose-6-phosphate, producing a cleavage to liberate inorganic phosphate ions. These ions of inorganic phosphate accumulate in sufficient amounts in specific areas to bring about a local supersaturation of the salt.[20] When the local supersaturation is achieved, there is a deposition of the mineral in the matrix. The necessary calcium for the formation of the bone salts is present in ionized form in the extracellular fluid[21] which circulates throughout the interstices of the bone matrix fibers.

The mineral salts of bone are stable within the matrix only at the normal tissue pH of 7.4. When inflammation tends to disturb the metabolism of carbohydrate in the cell, the result is an accumulation of lactic acid.

With the lowering of the hydrogen ion concentration in the localized area, the bone salts become increasingly more soluble. These salts go into solution, eventually leaving the matrix denuded of its mineral content. The roentgenographic picture of such a state shows a central area of rarefaction gradually diffusing toward a periphery of normal-appearing bone. Microscopically, the picture is one in which the bone cells appear unmasked and surrounded by a demineralized organic matrix.

It has been demonstrated in bone fractures that the pH will drop as low as 4.5, and lactic acid will accumulate in concentrations up to fifteen times that found in normal circulation.[15, 17] In such a low pH proteolytic enzymes become activated and reduce the organic matrix to solution.[22] It is assumed that this picture is precisely the same in the supporting bone of a tooth when the tissue is subjected to a localized irritant.

THE DEVELOPMENT OF THE LOCAL PERIODONTAL LESION

A local periodontal lesion is characterized by a progressive inflammatory involvement of the connective tissue, epithelium and alveolar bone. If the reaction is acute and severe, the periodontal fibers become edematous, and with an accompanying osteitis there is a greater degree of tooth mobility. When the condition is slowly progressive and chronic in nature, there is more often a definite marginal osteitis because of a fixation of the reaction so that the teeth are firm in their sockets even though a considerable amount of the supporting structure is lost. In this case the patient is not usually aware of the disease until it is in an advanced stage and an appreciable amount of damage has already been done.

Periodontal lesions of purely local origin have their beginnings in inflammation. The most common irritants producing gingival injury are calculus,[24] detritus, improperly placed restorations abnormal contours and positions of teeth, and bacterial toxins.

The local lesion may have its beginning in single or multifoci about the gingival crest. For example, an irritant may be present in either the mesial or distal interproximal areas, or less frequently, in the lingual, labial or buccal. It has been shown that the progress of the inflammatory process in the supporting structures of the teeth follows the course of the blood vessels.

The progress of the inflammatory reaction is, then, along the gingival perforating arteries to the interalveolar arteries and then to the periodontal membrane by way of the alveolar perforating arteries. It is for this reason that mild inflammatory processes can be persistent for long periods of time and have little effect on the periodontal membrane.[19]

The initial change in the periodontium is first observed in the connective tissue immediately underlying the epithelium where the capillaries penetrate into the connective tissue papilla of the gingival mucous membrane. The first imperceptible reaction probably occurs in the epithelium in the form of a nerve reflex. This reflex is conveyed to the lamina propria where it brings about a vascular change in the connective tissue papilla of the lamina propria. This marks the beginning of the inflammatory reaction.

With the development of the inflammatory reaction in the connective tissue overlying the alveolar crest, a characteristic change takes place in the surface epithelium of the gingival sulcus. This change may extend to the epithelial attachment of the tooth. The first reaction is a marked increase in the mitotic activity of the basal epithelial cells. If the inflammation is severe, mitosis is sometimes seen in the prickle-cell layer. When the degree of irritation is great, increased cellular activity may extend along the epithelium of the gingival sulcus to include the epithelial attachment. A marked increase in the production of keratin follows this increased cellular activity and a desquamation of the cells occurs in the sulcus and in the epithelial attachment toward the tooth surface.[18] The cellular desquamation adjacent to the tooth surface tends to deepen the gingival sulcus.

The connective tissue in immediate relation to the gingival sulcus is edematous. The collagen fibers are swollen and the nuclei of the connective tissue cells undergo pyknosis. Inflammatory cells mask the tissue elements. Because of the damage to the vessel walls, hemorrhage is almost spontaneous. The epithelium of the sulcus, as well as that of the crest, now appears to be ragged and unorganized.

The epithelial cells of the basal layer at the bottom of the sulcus and those attached to the tooth surface undergo a marked proliferation extending as convolutions toward the crest of the alveolar bone. These convolutions of the epithelium invade the connective tissue and break up the edematous gingivae and the gingival and transseptal periodontal fibers.[3]

As the inflammatory process extends along the gingival perforating arteries to the alveolar crest, the first roentgenographic evidence of bone involvement can be seen as a rarefaction of the cortical plate at the very tip of the alveolar crest. This may be interpreted as the establishment of a biochemical lesion with an involvement of the deeper structures. As the inflammation extends along the interalveolar arteries, the marginal alveolar crest begins to show, roentgenographically, an extending rarefaction. While the trabeculations of the supporting bone underneath the crest appear to be in varying degrees of demineralization, the cortical plate of the socket may remain intact until some time later.

The connective tissue of the alveolar crest and the transseptal periodontal fibers eventually go into solution and an open tissue lesion is evident. The periodontal membrane is edematous for a considerable distance down the side of the root. With the persistence of the irritant the inflammatory reaction is progressive, involving more and more of the periodontium until a definite pocket is established.

In the involved area there is initially a disturbance in the fluid exchange of the vessels of the connective tissue in the gingival crest. This is concomitant with a transitory increase in capillary pressure and a more permanent change in the vessel wall. The exudate is first serous; later fibrin occludes the vessels, and the lymphatics become thrombosed. With the impairment of the circulation, there is incomplete oxidation of carbohydrates due to an oxygen want which marks the beginning formation of lactic acid. The alkaline reserve of the tissue is depleted which, together with an increased glycolytic activity of the cells in the area, lowers the tissue pH and a local acidosis ensues. As the cell glycogen is exhausted, the energy source (pyruvic acid) is supplied by a breakdown of plasma proteins, and the area becomes a focus for a local gluconeogenesis. The cellular picture in a continued acidosis changes until all cells are destroyed and suppuration is the end result.

If this concept of the establishment of a local periodontal lesion by inflammation is accepted, then the periodontal lesion must be regarded in the same light as any type of lesion which involves connective tissue, epithelium and bone, and the biochemical processes and physical alterations which accompany inflammation must be considered identical in all tissues.

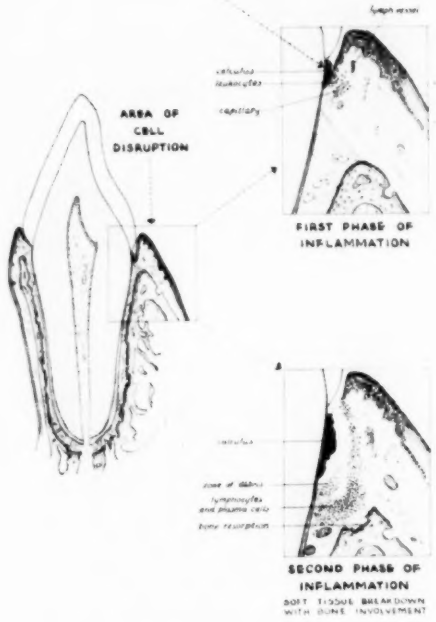
When the area of inflammation progresses to the "lesion stage," it becomes infected. The bacterial action then contributes to a more rapid progress of the disease by establishing a secondary irritant. The severity of the combination of these irritants determines the rapidity of tissue fixation. Tissue fixation reduces the inflammatory reaction to a subacute or chronic phase. A modification of the tissue pH and in the character of the cellular inflammatory elements is concomitant with this event. The shift in the cellular picture is outlined below:

Inflammation

Acute	Subacute	Chronic
Polymorphonuclear leukocytes		Plasma Cells
Mononuclear leukocytes	A combination of acute and chronic types	Lymphocytes

The transition from the acute to the chronic phase is accompanied by character-

THE PERIODONTIC LESION



NERVOUS REFLEX
probably occurs too early for determination

→ Vasoconstriction

LEUKOTAXINS
from injured cells

→ *Vasodilatation and congestion*
Increased capillary permeability
Increased flow of plasma proteins
Migration of leukocytes
Fibrinogen converted to fibrin by contact with injured tissue. This walls off the site of irritation.
Clotting also occurs in the neighboring lymph vessels producing lymphatic blockage.

NECROSIN
possibly a proteolytic enzyme from the euglobulin fraction of the exudate which produces further cell injury and possibly precipitation of fibrin

→ *Edema from increased plasma flow and vascular blockage*
Anoxemia
Loss of CO_2
Beginning gluconeogenesis
Incomplete glycolysis
Formation of lactic acid instead of CO_2 and H_2O
Drop in pH

Other biochemical agents not considered here are the leukocytosis promoting factor, the chemical basis of fever, and the leukopenic factor in inflammation.

Healing at this point does not necessarily involve actual tissue breakdown and formation of pus.
There may be no apparent bone involvement at this time.
Repair may involve an ingrowth of fibroblasts and new capillaries.

GLUCONEOGENESIS + ANOXEMIA
formation of lactic acid; lowering of pH.

→ *Continued lowering of tissue pH due to anaerobic cell respiration with production of lactic acid.*
Polymorphonuclear leukocytes are destroyed leaving macrophages.
Further increase in acidity.
Macrophages are destroyed.
Bone perforation and frank pus results with pocket formation.

Healing is accomplished by removal of irritant, removal of toxic material, establishment of basal cell, fixation of tissue.
There is an ingrowth of fibroblasts, new capillaries appear and a remineralization of the bone matrix occurs.

istic changes in the physical appearance, both microscopic and macroscopic, of the tissues of the periodontium. Over a long period of time there is a progressive resorption of the alveolar bone and a concomitant deepening of the pathological pocket. The alveolar bone in the chronic phase differs from the acute in that what was seen as a diffuse rarefaction becomes a rather definite marginal rarefaction which in many cases seems to be walled off from the normal-appearing bone directly underlying the more superficial area of active resorption. Paralleling the microscopic changes, the physical alterations are a change in the color of the tissue from the rubor of acute inflammation to the bluish-red appearance of stasis; there is an atrophy and a fibrous appearance of the gingiva and a deepening of the gingival sulcus.

The inflammatory picture of periodontitis is rarely of violent nature. The onset is generally so gradual as not to be detected by the patient and far too often not by the dentist, the exception to this being an acute ulcerative gingivitis.

In the chronic condition the impaired circulation leading to stasis is probably the persistent factor in the maintenance of a disturbed metabolism, for the oxygen supply to the tissue is impaired. This again points to a stereotyped picture of inflammation which has as its basis a disturbed cell physiology and a change in tissue pH. The results are a breakdown of connective tissue and epithelium and a resorption of the alveolar bone. A chronic low-grade inflammatory process may persist for a long period of time. There is just enough disturbance of cell metabolism to produce a slow and continuous destruction of the periodontium. In a picture of this kind there is often little of a clinical nature that indicates a systemic factor, although it is recognized that certain metabolic disturbances contribute to the overall picture.

From the foregoing discussion, the development of the periodontal lesion should be looked upon in the same manner as any surface lesion in which connective tissue, epithelium and bone are involved. Inflammation in either case is the pivotal reaction in the response of tissue to injury. The mouth and its peculiar tissue relationships only demonstrate this more subtilely. In the periodontal lesion the progress of the inflammatory reaction to the extent of tissue liquefaction usually produces an active resorption in the underlying bone structure. A pathological pocket indicates that tissue degeneration in the localized area has progressed to the point of destruction of the periodontal fibers from the tooth surface.

The Sequence of Events in the Formation of the Pathological Pocket Resulting from a Local Injury

Primary irritant—calculus—trauma

↓

Inflammation
1. Progressive involvement of lamina propria
2. Epithelium
3. Bone

↓

Periodontal tissue breakdown with beginning pocket formation.

↓

Infection as the secondary irritant

↓

Rarefaction of the alveolar crest

↓

Definite pocket formation

↓

Persistent chronic inflammation with calculus, detritus and bacterial toxins constituting the irritating factors. Progressive extension of the pathological pocket with rarefaction and resorption of the alveolar crest due to a lowered pH over a long period of time with underlying supporting bone appearing normal. Mobility of teeth accompanying the late stages of the disease.

THE ESTABLISHMENT OF THE PERIODONTAL POCKET

An injury which has been severe enough to produce tissue loss and the formation of a pathological pocket heals by forming granulation tissue. As in larger wounds extending into deeper structures, repair involves the partial filling in of the defect and the establishment of epithelial continuity. In the periodontal lesion, following the removal of the irritant, as in healing "by secondary intention," there is a partial replacement of the connective tissue through proliferating gran-

Sequence of Events in Pathological Pocket Formation Resulting from Systemic Dysfunction with the Local Irritant Acting in a Secondary Role

Systemic dysfunction (Example: Vitamin C)

↓

Incomplete maturation of extracellular matrix resulting in the breakdown of tissues of the periodontium.

↓

Insidious mobility of affected teeth which, together with other local disturbances, superimpose an injury on a tissue of lowered resistance.

↓

Marginal tissue breakdown, local disturbed metabolism, infection, pocket formation.

↓

Extreme mobility of teeth accompanied by a marked breakdown of all of the tissues of the periodontium.

ulation tissue. The affected part has a tendency to undergo organization and repair but not regeneration to the original form. If there has been a reduction in the alveolar crest, there may follow a concomitant reduction in the associated structures, and the root of the tooth is exposed.

Healing of a periodontal lesion, as described above, *where there has been no surgical interference,* involves the development of a connective tissue stroma in the soft-tissue side of the lesion. This stroma, known as the granulation tissue, fills the defect in the wound surface. This tissue, when seen from the surface, is first composed of small granules which give the tissue its name. Each granule is composed of a central vascular core with fibroblasts and other cells in the intervascular spaces. The fibroblasts serve as adventitia for the sprouting capillaries, and as the tissue matures, collagen is deposited. (Granulation tissue of normal repair should not be confused with the excessive production of granulation tissue—granulomatous tissue—which results from a long and continued chronic inflammatory process.) In response to the marked tissue destruction along the side of the root, epithelial cells proliferate inward from the periphery of the wound. The advancing epithelium has the appearance of unorganized embryonic epithelium. With organization there develops a basal layer which becomes attached to the primitive connective tissue below by a basement membrane. These cells gradually organize into stratified squamous epithelium and form a lining of the wound surface as far as the connective tissue attachment to the root. *The periodontal pocket has now been established!* Prior to this time the area has been a tissue lesion, and tissue repair in the periodontium is essentially the same as in other lesions which involve similar tissues.

Thus the epithelium of the gingival sulcus does not creep down the root surface, but organizes to form a stratified squamous layer covering the connective tissue. When a definite pocket exists, there is no way by which the granulation tissue of repair can bridge to the surface of the tooth and become attached to it, because an epithelial lining intervenes. There can be no fibrous repair in the form of a periodontal membrane without surgical intervention to remove the epithelial lining and to initiate a blood clot. The important point is that in periodontal pocket formation, *epithelium does not creep or proliferate down the surface of the tooth,* but proliferates from the gingival surface epithelium to cover the wound surface and form the epithelial lining of the pocket.

Although a very slow gingival recession is less dynamic in action, it may be envisioned to operate in a similar way. The processes are of greater subtlety and may not be observed clinically except for their final result. It is not possible to detect them from histological examination and, therefore, they must be reduced to the biochemical equation. It is not necessary to have a "full-blown" inflammatory reaction to produce tissue changes. An anoxia (oxygen want), due to faulty circulation in the part, may bring about an abnormal physiology. There are certain imperceptible changes, such as degenerative change accompanying old age,[24]

(Continued on page 44)

Case Report

Eosinophilic Granuloma Diagnosed by Gingival Biopsy

By HAROLD E. GRUPE, D.D.S., *Amarillo, Texas*, AND
BALINT ORBAN, M.D., D.D.S., *Colorado Springs, Colorado**

INTRODUCTION

EOSINOPHILIC granuloma is a disease of the bones. It is a destructive but benign disease. The correct diagnosis is of great importance, since eosinophilic granuloma can be cured by conservative surgery as well as by roentgen radiation. The authors of this report do not know that eosinophilic granuloma ever has been diagnosed by gingival biopsy. Most cases reported in literature were diagnosed by biopsies taken from bones.

LITERATURE

The eosinophilic granuloma was first described in 1940, almost simultaneously by Otani and Ehrlich[1] and Lichtenstein and Jaffe.[2] The latter authors describe this disease in a second article[3] as a "Condition affecting one, several, or many bones, but apparently limited to the skeleton, and representing the mildest clinical expression of the peculiar inflammatory histiocytosis, Letterer-Siwe disease and Schüller-Christian disease." "In the cases which we reported, we found the lesion to be characterized histologically by conspicuous sheet-like collections of histiocytes (including some revealing phagocytic activity), interspersed among which there were more or less prominent accumulations of eosinophilic cells and especially eosinophilic leukocytes. In addition, however, we usually saw fields of hemorrhage and necrosis and, particularly in relation to these fields, larger or smaller numbers of multinuclear giant cells, some of which likewise showed phagocytic activity."

The authors add that "In eosinophilic granuloma of bone, the lesions seem to develop only in the skeleton," and they believe that this disease is "a peculiar inflammatory reaction to some, as yet unknown, infectious agent."

In the dental literature, a number of cases have been reported. The most complete clinical, as well as histopathologic description, was given by Kurt H. Thoma,[4,5] giving also details about the roentgenologic therapy. Others who observed such cases are: Bailey, J. W., and Freis, E. D.;[6] Salman, and Darlington, C. G.;[7] Cox, M. E.,[8] and lately, Kruger, G. O., Prickman, L. E. and Pugh, D. G.,[9] from the Mayo Clinic. These authors give an excellent review of literature and discuss the possibilities of connections between the benign eosinophilic granuloma with some forms of xanthomatosis. Reference is also made to the book on "Bone and Bones," by Weinmann, J. P., and Sicher, H.,[10] who discuss this disease under the heading of "Inflammatory Reaction of the Reticular Elements."

CASE REPORT

The case to be reported here is that of a woman twenty-six years of age. Gingival disturbance started about four years previously as a marginal, ulcerative condition around the upper left first molar and lower left first and second bicuspids with considerable bone resorption between the two bicuspids, (Figure 1). The bicuspids were extracted about three years ago and the gingival condition improved, but now is manifest on the lower left cuspid and lateral incisors, extending to the distal surface of the cuspid. Medical history shows that this patient was confined to a hospital four years ago with pulmonary fungus infection and fungus infection of the ears, (aspergillus nigrans). Recent chest roentgenograms and physical check-up revealed chest condition

*From the Colorado Dental Foundation, Colorado Springs, Colorado.

Fig. 1. Roentgenogram of lower bicuspids, showing bone resorption, similar to that seen in cases of periodontosis.

apparently inactive, but treatment of the ears is continued one to two times a week with only slight improvement.

The gingiva at the time of the present examination was edematous, with slight necrotic areas at the gingival margin around the left lower cuspid and lateral incisor. Similar changes appeared on the lingual side of the upper incisors and lower right first bicuspid. The roentgenogram taken at this time (Figure 2), reveals healing of the sockets of the extracted bicuspids, but in one area a slight porotic area can be seen. A biopsy specimen was taken from the interdental papilla between the lower left lateral incisor and cuspid, extending to the labial and distal surfaces of the cuspid.

HISTOLOGIC FINDINGS:

The specimen was fixed in a 10% neutral formalin solution, embedded in paraffin, stained with hematoxilin-eosin. Sections were cut in labio-lingual direction. Of special interest is the area just distal of the cuspid, (Figure 3). The labial surface of the gingiva is covered by epithelium, which is unhornified. The border between epithelium and connectvie tissue is irregular, and does not show the usual epithelial ridges. In some areas, the epithelium is rather thin; in others, somewhat heavier. The tip of the gingival papilla is also covered by epithelium which can be followed into the pocket epithelium without interruption. The lamina propria of the mucous membrane appears densely infiltrated by cells which in low magnification, appear in some areas rather pale; in others, darkly stained, (Figure 4). The pale cells are often surrounded by the darkly stained cells. In high magnification, the dark cells can be recognized as eosinophile cells, (Figure 5). In certain regions, these cells are very numerous and densely

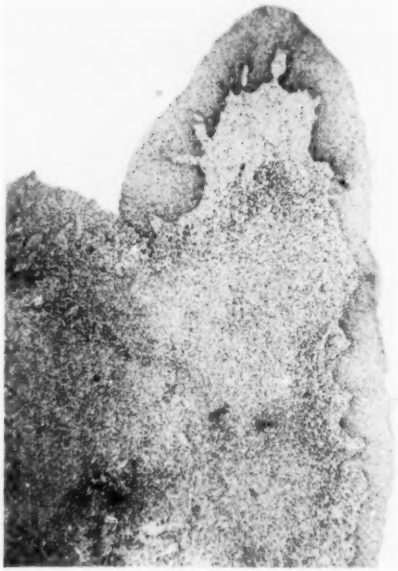


Fig. 3. General view of gingival biopsy from distal surface of cuspid. Entire gingiva densely infiltrated by large pale cells—interspersed by darkly stained cells.

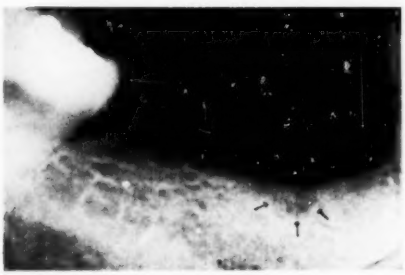


Fig. 2. Roentgenogram of area seen in Figure 1 three years after extraction of bicuspids. Rarefied area indicated by arrows.

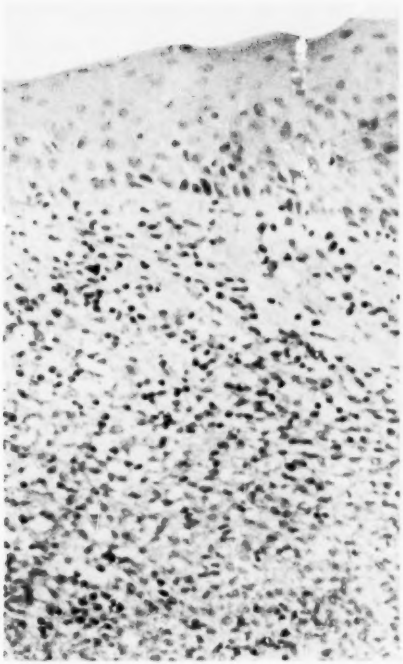


Fig. 4. Higher magnification of gingiva. Epithelium thin, without hornification. The lamina propria is densely infiltrated and shows no papillary layer. Areas of pale cells alternate with darkly stained cells.

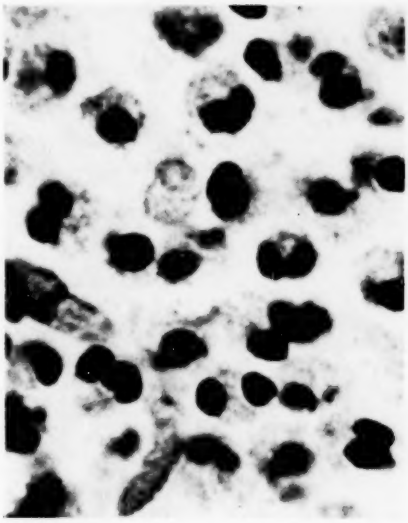


Fig. 5. High magnification of darkly stained cells —eosinophile leukocytes.

arranged. In other regions, they are dispersed between the lightly stained cells. These are histiocytic cells, (Figure 6), with large, irregular nuclei with finely distributed chromatin. The nuclei appear round or kidney-shaped, frequently as if folded. Only very few plasma cells and lymphocytes appear among these histiocytes, but there are certain areas where lymphocytes are quite numerous. Cells are frequently found in different stages of mitotic division, (Figure 7). In some areas, the cytoplasm of the histiocytes appears foamlike, resembling xanthom cells. They are most probably histiocytes laden with lipoids, (Figure 8).

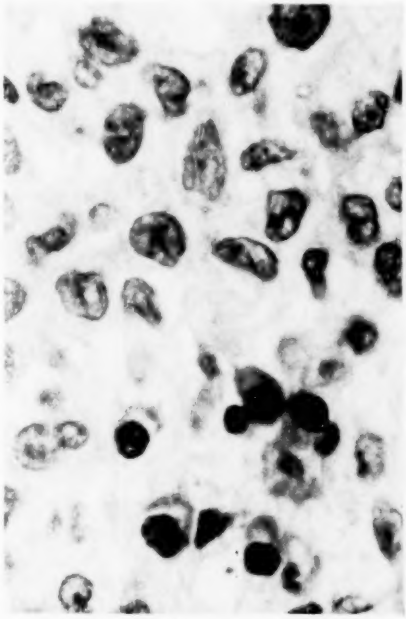


Fig. 6. High magnification of pale cells. Histiocytes with large cytoplasm, pale, irregular nuclei. Few plasma cells.

DISCUSSION

The findings are typical for eosinophilic granuloma. The presence of an eosinophilic granuloma in the gingiva should not be taken

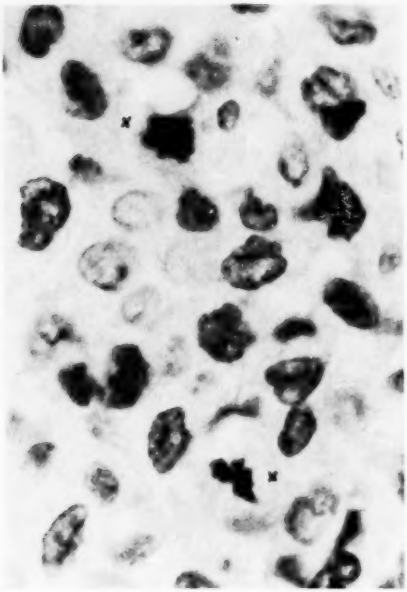


Fig. 7. Mitosis (X) of histiocytes.

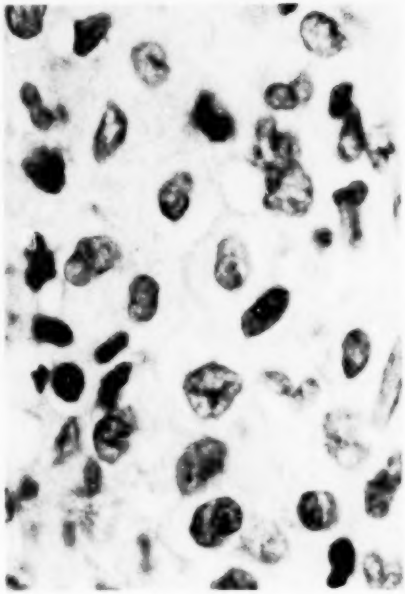


Fig. 8. Xanthom cells (foam cells). Histiocytes laden with lipoids, (phagocytes).

as evidence that this disease can have an extraosseous origin. The focus may well have developed in the marrow spaces of the alveolar process and continued to exist after the extraction of the teeth, in the rarefied area, (Figure 2), and may have invaded the gingiva secondarily. A similar pathogenesis is assumed for the giant cell "epulis" by Weinmann and Sicher[10] who consider this disease as identical with the intraosseous brown tumors, or giant cell nodes.

The one unusual finding in the present case is the presence of the "xanthom" cells. A similar finding was presented by Kruger, Pickman and Pugh,[9] and they expressed their opinion that this finding might indicate a certain relationship between eosinophilic granuloma and xanthomatosis, as was also assumed by Lichtenstein and Jaffe.[2] Weinmann and Sicher[10] report in this respect: "Three diseases are believed to be of the same principal type, namely: Letterer-Siwe's disease, Hand-Schüller-Christian's disease, and the eosinophilic granuloma." Also, "Letterer-Siwe's disease seems to be acute and more generalized; Hand-Schüller-Christian's disease, a chronic and more localized type of the same lesion. Their close relation is made probable by the observation that the lipidization and the scarring which are so characteristic for Hand-Schüller-Christian disease have also been found in the rare instances of subacute Letterer-Siwe's disease after duration of one to two years. The lipidization is caused by the phagocytosis of cholesterin by the histiocytes which then assume a more or less typical appearance of foam cells or xanthoma cells."

The occurrence of the xanthoma cells as observed by Kruger, Pickman and Pugh, as well as in the case presented here, seems to bring these three diseases even closer together. They seem to be a somewhat different manifestation of the same disease complex, histiocytosis.

When the oral symptoms first occurred,

the patient suffered from a fungus infection and this infection is still not quite under control. The question of a possible etiologic connection might be taken into consideration, especially in view of the opinion of Lichtenstein and Jaffe,[2] that this disease is "a peculiar inflammatory reaction to some, as yet unknown, infectious agent."

No progress of bone destruction has been observed since the bicuspids were extracted. No other bones of the skeleton have shown rarefied areas. However, a few months ago, the patient developed an acute otitis media and mastoiditis. A mastoidectomy was performed. The post-operative course did not follow the expected favorable progress, and upon the basis of oral biopsy findings, the mastoid wound was re-opened and a biopsy taken. The pathologist's report was "eosinophilic granuloma with super-imposed chronic inflammatory reaction." Roentgen therapy was recommended and carried out. Patient is improving, and the case will be kept under observation.

SUMMARY

An eosinophilic granuloma was diagnosed by microscopic examination of a gingival biopsy specimen. The clinical features of the disturbances observed in this patient's supporting tissues were at some previous time those of a periodontosis, (Figure 1), and some ulcerative gingivitis of a mild and chronic character. Microscopic examination of biopsy specimens gains considerable importance through this observation, not only as far as the oral condition is concerned, but the general well-being of the patient, as well.

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The Role of Nuclear Physics in Medicine and Dentistry

By D. H. COPP, M.D., *Berkeley, California*[1]

THE twentieth century has seen a tremendous development in the field of nuclear science, which began with the discovery of radioactivity by Becquerel in 1896 and reached a climax with the development of the uranium pile and the atomic bomb in 1945. Medical science has been quick to apply these discoveries. Three years after radium was first isolated by the Curies in 1898, it was being used to treat skin disease and cancer. However, the chief contribution came with the artificial production of radioactive isotopes of the common elements. This was first accomplished by Curie and Joliot in 1934, using a radium-beryllium source to produce radioactive phosphorus. The development of the atom-smashing cyclotron by E. O. Lawrence in 1936, and the chain reaction pile in 1942 greatly increased the number and quantity of these radioactive isotopes available for medical use.

These isotopes are atoms with the same nuclear charge and chemical properties as the

1. Division of Physiology, University of California, Berkeley.

Shortened version of paper presented to the Thirty-Third Annual meeting of the American Academy of Periodontology, October 14, 1949. San Francisco, California.

TABLE I.

Properties of Radioactive Isotopes Important in Medicine and Dentistry

Element	*Isotope*	*Half-life*	*Radiation*	*"Safe Tolerance Dose" (70 Kg. man)**	*Cost°*	*Important Uses*
Phosphorus	P^{32}	14.3 days	beta–1.69 mev.	0.16 mc	$1.10 per mc	(1) Metabolic studies (2) Mineral exchange—bones and teeth (3) Localization of tumors (brain, breast) (4) Treatment—leukemia, polycythemia (5) Treatment—skin cancer (6) "Tag" red cells—blood volume detn.
Iodine	I^{131}	8 days	beta–0.6 mev. gamma–0.367 mev. 0.080 mev.	0.0002 mc	$1.00 per mc	(1) Studies of thyroid metabolism (2) Diagnosis of thyroid disease (3) Treatment of hyperthyroidism (4) Treatment of thyroid cancer (a few cases)
Sodium	Na^{24}	14.8 hrs.	beta–1.4 mev. gamma–1.4 mev. 2.8 mev.	0.090 mc	$12 per 15 mc	(1) Absorption studies (2) Measurement of extracellular fluid (3) Measurement of circulation rate (4) Detn. of efficiency of peripheral cir.
Iron	Fe^{55} Fe^{59}	4 yrs. 44 days	gamma–0.07 mev. beta–0.26, 0.46 mev. gamma–1.1, 1.3 mev.	0.075 mc	$33 per 1.9 mc	(1) Study of iron metabolism and anemia (2) Absorption of iron (3) "Tagged" red cells—a) blood volume b) survival of stored blood
Calcium	Ca^{45}	180 days	beta–0.25 mev.	0.070 mc	$2.20 per mc	(1) Study of metabolism of bones and teeth
Strontium	Sr^{89}	55 days	beta–1.5 mev.	0.028 mc	$1.35 per mc	(1) Study of metabolism of bones and teeth
Fluorine	F^{18}	112 min.	beta–0.7 mev.		(cyclotron only)	(1) Skeletal studies; fluorosis
Carbon	C^{14}	5100 yrs.	beta–0.154 mev.		$36 per mc	(1) Metabolic studies
	C^{11}	20 min.	beta–0.95 mev.		(cyclotron)	(1) Photosynthesis

*Values for "safe tolerance dose" for a 70-Kg. man are from Marinelli, Quimby and Hine(1).

°Prices are quoted from Catalogue & Price List No. 3, Isotopes Branch, U.S. Atomic Energy Com., Oak Ridge Tenn. (revised to 1949).

normal element, but with an unstable nucleus, which disintegrates with the emission of radiations. Since these radiations may be easily measured with a Geiger counter or other similar device, it is possible to detect the presence of the radioactive isotope wherever it may go in the body. Such "tagged" elements have proven invaluable in studying the function of the body in health and disease.

While chiefly of use as radioactive tracers in fundamental medical research, radioactive isotopes are bieng used increasingly in the diagnosis and treatment of certain diseases. In addition, the radiation hazards involved, and the threat of the atomic bomb has focussed attention on the new problems of radiation medicine.

RADIOACTIVE ISOTOPES

The fundamental unit of radioactivity is the Curie, which is defined as the activity of radon gas in equilibrium with 1 gram of radium. A smaller unit, the millicurie (0.001 Curies) is more commonly used, and this is defined as the amount of radioactive isotope in which 3.7×10^7 disintegrations occur per second.

There are several factors to be considered in selecting an isotope, particularly for clinical work. The isotopes available from the Isotopes Branch of the U.S.A.E.C. are listed in their catalogue. Total shipments exceed 5,000. Recently, the commission has agreed to supply isotopes for use in cancer research and treatment at no cost except for handling charges. A prime consideration in choice of isotope is the half life, or time during which half of the radioactive atoms present decay. It is usually not feasible to carry on an experiment for longer than 5 half lives (at which time only 3% of the original activity remains). For this reason, isotopes with half life of less than 12 hours cannot be used for chronic experiments, and are inconvenient even for acute experiments. On the other hand, there is a reluctance to use radioactive isotopes with a half life longer than 2 months in human beings because of the possible harmful effects of long continued chronic radiations.

A list of the radioactive isotopes commonly used in medicine and dentistry is given in Table 1, along with the cost and certain important properties. The most widely used have been radiophosphorus (P^{32}), radioiodine (I^{131}), radiosodium (Na^{24}), and radioiron (Fe^{55}, Fe^{59}).

HEALTH PHYSICS

Although radioisotopes have been handled safely by thousands of individuals, they are potentially dangerous substances. The toxic effects of x-rays are well known, and it has been recommended by radiologists that exposure to these radiations should not exceed 0.1 roentgen per day. Similar precautions are necessary with the radiations from radioactive isotopes. In addition, special care must be taken to prevent these toxic substances from getting into the body, since the tragic fate of the radium dial painters has demonstrated the fatal effects which may be produced. Such contamination can usually be prevented by following the general rules of surgical asepsis.

Because of these hazards, work with radioactive isotopes has generally been restricted to individuals with previous experience, and to institutions equipped to supervise these activities.

"SAFE TOLERANCE DOSE"

In addition to the above precautions, special care must be taken to safeguard the welfare of the patient when these substances are used clinically. Unless the radio-isotope is being used for the therapeutic effect of its radiation, or there are other extenuating circumstances, the amount administered should not exceed a "safe tolerance dose." This has been defined by Marinelli, Quimby and Hine[1] as the amount which will deliver not more than 0.1 roentgen equivalents of radiation to any tissue of the body during the first 24 hours following administration. The "tolerance" values for the commonly used isotopes are given in Table 1.

APPLICATIONS IN MEDICINE

Less than 15 years have passed since George Hevesy used the first artificially pre-

pared radioactive isotope (P^{32}) to study phosphorus metabolism in the rat. Since that time, the use of isotopes has become widespread, both in fundamental research and in clinical medicine. Over a hundred medical centers, universities and other institutions are actively engaged in isotope work, and in at least half of this number, there is considerable clinical use in the study, diagnosis and treatment of disease. A number of excellent reviews[2-5] are available, and attention is directed to the fine monograph by Dr. H. V. A. Low-Beer[6] which is soon to be published.

APPLICATIONS IN DENTISTRY

Although the advantages of radioactive isotopes in studying certain dental problems has been stressed by McCauley in his excellent review,[7] the actual work has been very limited. Much of this work has been concerned with the turnover of mineral in normal and diseased teeth, and with certain problems of skeletal metabolism.

Hevesy, in his first experiment with radiophosphorus in the rat, observed a marked uptake of P^{32} in bones and teeth, including some turnover of phosphorus even in the enamel. Unerupted teeth and the growing incisors of the rat showed a much greater uptake than did the mature molars.

The mechanism governing this mineral exchange was studied by Hodge et al.[8-10] who found that finely ground ash of bone, dentine, enamel and apatite all absorbed radiophosphorus, radiostrontium and radiofluorine according to physical principles, the curves following the Freundlich isotherms. Such absorption could account for the uptake of mineral by mature skeletal tissues; it does not, however, account for the greater uptake in areas of bone or tooth growth.

The relative uptake of P^{32} in various parts of the tooth were studied by Volker and Sognnaes,[11] who observed almost twice as much P^{32} in root dentine as in the coronal dentine, while there was very little in the enamel. By blocking the various parts of the tooth, Barnum and Armstrong[12] were able to determine the route of penetration of radiophosphorus. While most of the P^{32} came through the pulp, considerable amounts were able to penetrate through the cementum. Very small but definite amounts were also able to get through the enamel.

Wassermann et al.[13] studied the same problem in vivo in dogs. If the pulp was destroyed, or the canal was blocked, absorption of P^{32} was reduced 90%. The residual penetration (10%) presumably occurred through

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the cementum. This amount was further reduced in cases of periodontal disease.

Similar findings were obtained in human patients.[14] When exposed to mouth wash solutions of phosphate containing P^{32}, very little radiophosphorus penetrated the enamel. However, when a cavity was present, considerable amounts of P^{32} penetrated to the dentin through the carious area.

Despite the interest in fluorine, the only isotope available—F^{18}—has a very short half life (112 minutes) and so its use has been limited. Volker, et al.,[15] found definite amounts of F^{18} taken up by dental tissue even after only 35 minutes. Largest amounts were found in the root dentine and in the growing areas of the rat incisors. Because of the interest in topical fluorine applications to teeth, it would be interesting to study the uptake of F^{18} by normal enamel, and by carious areas.

CONCLUSIONS

Although still limited in their use, radioactive isotopes have already attained a real and important place in clinical medicine. They should also prove useful in dental research because they provide one of the few methods for studying the limited metabolic activity of bone and tooth structures. The ease with which minute amounts of these radioactive materials can be accurately measured and distinguished from the mass of inert mineral in the tooth is particularly valuable. They should find application in studying calcification, mineral exchange, fluorosis, carious changes, periodontal disease, root resorption, dental nutrition and many other related problems.

Presentation of Dental Services

By A. J. PEETZ, D.D.S., *Madison, Wis.*

WHEN the "depression" of 1933 swept the country it not only found the business and commercial world unprepared but also affected the professional world to the extent that it found many members so unprepared that they were forced to apply for government relief or work on W.P.A. Since a shortage of dentists does exist and as dentistry is a necessity, can we avoid fluctuations in our offices which were so prevalent in the time of our last "depression" which started with the stock market crash in 1929? There is no doubt that 1948 was the last of the bonanza years. Not only dentists but all business men forty years old or older should not expect to see the like of that again.

It was about 1929 that an article was published in one of our dental journals by Dr. George Clapp, of New York, who had made a study of the reasons why some dentists are successful and others are not, although practicing adjacent to one another in the same building. At that crucial financial period he made a survey of five thousand dentists and stated that even during normal times a certain group was unsuccessful while another group was able to retain a practice to the extent that they still were able to buy new equipment and maintain beautiful homes. After an intensive study of why these men were successful in spite of a depression he discovered that they were interested in preventive dentistry.

Feeling that there must be some solution of the loss of patients at the time of reduced incomes, therefore, after a great deal of deliberation and study I worked out a Yearly Preventive Plan. This plan has been in operation for twenty-one years and it has proved to be the most workable method that can assure the patient that he will minimize his dental troubles as well as the possibility of never having to wear dentures. The

Presented at the Thirty-Third annual meeting of the American Academy of Periodontology, October 15, 1949, in San Francisco, California.

Preventive Plan is a yearly service which consists of complete mouth x-rays every year, a preventive treatment every four months (and we do not use the term "prophylaxis" or "cleaning"), a toothbrush demonstration and a continual supervision of same. This service is performed for a stipulated fee to be paid at the time the x-rays are taken.

The first few years the plan was in operation it was optional whether the patient desired to go on this plan or not, but in the last ten years I have made it compulsory. If they do not subscribe to the plan I do not accept them into my practice. I feel that if a patient is not interested enough to invest a small amount of money for preventive dentistry then he is not a good patient and will eventually drift elsewhere. This plan does not apply to students, whom we do not encourage in our practice, or a transient patient. In the twenty-one years that I have had this plan in effect in my office I have proven two things:

1. The patient who is on this plan has a beautifully clean mouth and

2. The immaculate care that is given minimizes the chance of loss of teeth. If a mouth is radiographed yearly and receives a minimum of three preventive treatments, or more as you wish, a year as well as proper toothbrush supervision, there is very little chance for any oral breakdown, unless a patient has some serious systemic disturbance. My point in having them pay the fee for this plan in advance is that they will come in regularly when notified.

If this phase is neglected and it is up to the patient to make an appointment every four months and pay for the treatment as it is given, few of them will report regularly. Also if they were to pay for x-rays individually and for the preventive treatments every four months the cost would be prohibitive to the average patient. By paying in advance for the plan he will keep his appointments and receive the benefit of that for which he has already paid. The reaction of the public to this program has been very favorable as expressed by two professors at the University of Wisconsin, who subscribed to this plan about eighteen years ago. In both cases their statement has been, "if every dentist in the United States would operate such a plan, your profession would never have to worry about panel or socialized dentistry."

The Preventive Plan is only practicable when the dentist has a full-time hygienist in the office. Since many states now license hygienists, to employ a girl this work is not only effective, but highly profitable. It has been my experience not to permit the hygienist full responsibility as far as treatment and toothbrush technique is concerned. If a patient is on the plan for several years and is not following through with the instructions given him, he is again given an appointment with the dentist who will repeat the instructions with him as well as to inform him that it is his responsibility to carry on.

Dentists' greatest trouble is to be too anxious to begin work without having the proper understanding of the conditions that exist in the mouth. By conscientiously working such a plan one becomes more interested in the surrounding tissue and has a better understanding of the patient's wants. Unless this preventive service, which is a well defined, efficient plan, is functioning from the beginning to the end of each case it will not be very successful. It is hard to understand why so few members of our profession do not recognize when the tissues show a marked deficiency. The companies who are in the commercial field advertise such slogans as "four out of five have it," etc. until the people are beginning to recognize that when gums are bleeding or inflamed that they need attention. Yet in many cases the patient will state that he has been to his dentist and all the information he was able to get was to brush harder, or he was getting older and would soon lose his teeth anyway. Nothing was done to instruct the patient as to what could be done to improve this condition of which he himself was conscious. With such a circumstance it is not hard to understand why dentists are not held in esteem by the

medical profession and the public as well, and are referred to as "tooth mechanics." This proves the point that members of our profession are too interested in the mechanical side of dentistry. Dentists will lose a real opportunity if they do not devote some time to improving oral conditions as well as teaching the patients their obligations to their mouth condition. By putting in some form of preventive plan one definitely can recognize conditions of this type in their incipiency, and as soon as the dentist recognizes this condition and remedies it, he gains the confidence of his patient. Many patients leave dentists because of the laxity shown regarding proper care of both gums and teeth and the haphazard methods used when making a diagnosis. Since so many dentifrice companies are educating the public to the importance of seeing their dentists because of bleeding gums, we should cooperate to the point of at least recognizing and instructing them as to the dangers of this condition.

Toothbrush technique is demonstrated with a large model and brush, as well as having the patient bring his toothbrush to give you the opportunity to see what type of toothbrush he uses and how he cares for the brush.

After the patient has subscribed to the plan and has had his x-rays taken, a second appointment is given for the diagnosis of the x-rays and a complete survey of the mouth. A chart is made of the previous work which has been done, also some pertinent information as to the age of the patient, condition of the gums, condition of the teeth, abnormalities of occlusion, etc. A third appointment is then given for estimates and recommendations of the work to be done to restore his mouth to a healthy condition. On this appointment the patient is likely to be apprehensive and nervous as he expects the worst. It is well to take this into consideration and calm his fears right in the beginning. Never say "we have bad news for you," even if the only solution to his problem is to have all teeth extracted. Explain in this case he has a fine ridge to work on and that he is very fortunate because with modern dentistry, replacements can be made comfortable and beautiful. It is advisable to show the patient the x-rays, explaining in detail what is to be done and the condition of the mouth.

At the time the appointment is made for the patient to receive recommendations and estimates always impress them they are not investing in inlays, amalgams, or synthetics, but they are investing in dentist's time and skill. Never mention materials, since doing that creates shoppers. It would be much better that dentists impress upon them that the investment is in dentist skill and time. Since some patients are not in a financial position to have a completely rebuilt mouth, a program including the most necessary work, be it either restorative or periodontal treatments should be suggested with the recommendation that other work be taken care of at a later time.

THE AMERICAN BOARD OF ORAL PATHOLOGY

The American Board of Oral Pathology will conduct its next examination at Atlantic City, New Jersey, in conjunction with the 1950 meeting of the American Dental Association. The exact date, time and place will be announced at a later date. The examination will be both practical and oral. Individuals who contemplate appearing before the board should make arrangements to submit their credentials as soon as possible.

Prospective candidates may secure detailed information by addressing the Secretary, Lt. Colonel Joseph L. Bernier DC, Armed Forces Institute of Pathology, Seventh Street and Independence Avenue, SW, Washington 25, D. C.

Are Rampant Dental Caries and Periodontal Disease Incompatible?

By ROBERT G. KESEL, D.D.S., M.S., *Chicago, Ill.*

THE answer to the question raised in the title of this paper is not an easy one. An incompatibility or even an antagonism between dental caries and periodontal disease has been stated numerous times but a review of the literature has failed to reveal valid proof for the contention. Clinical reports on the simultaneous activity of these two diseases are conflicting, and are based largely on personal impressions rather than supporting evidence. The consensus seems to be that there is an antagonism which is based on the belief that caries results from fermentation and periodontal destruction from proteolytic and putrefactive processes and that these two reactions do not occur simultaneously in the immediate environment of a tooth.

There is not even agreement on a correlation between gingivitis and dental caries, even though gingivitis may be a forerunner of periodontal destruction and may result from mechanical irritation produced by the carious lesion and not from some chemical or bacteriological condition that is common to both.

Only a few studies were found in which an attempt was made to establish a quantitative relationship between caries and gingivitis. In 1913 G. V. Black[1] compiled data from 500 persons who ranged in age from twenty to thirty-five years. Ninety-five per cent had some degree of gingivitis. About 5 per cent were due to carious cavities on the axial tooth surfaces, 18 per cent to bad margins on restorations, 10 per cent to lack of contact and 7 per cent to improper contact of restorations. Approximately 40 per cent of the gingivitis was associated with the carious process or its repair, but it must be emphasized that the gingivitis resulted from the previous activity of caries and does not constitute proof that the two processes were active side by side at the same time.

About four years ago Shay and Smart[2] reported indications of a positive correlation between caries and gingivitis. They examined ninety-eight airmen and found that two-thirds of those with caries had gingivitis while only one-third of those with no active caries had gingival inflammation. They offered statistical analysis to show that the difference was significant and they concluded that gingivitis was more prevalent in a carious mouth.

Day,[3] in 1944, and King,[4] in 1945, on the other hand, reported no relationship between caries and gingivitis. Day examined 200 malnourished boys averaging 14 years of age in the Kangra Valley of India and observed a remarkably low incidence of caries. Thirty-six per cent of the subjects had no caries; the others averaged one and a half cavities per child and a high percentage of the lesions recorded were defective fissures not actual cavities. Gingivitis, however, was very prevalent; 81 per cent showed from slight to very severe conditions. He concluded that the high incidence of gingivitis in these children had no relationship to dental caries.

King reported on 387 persons ranging in age from 12 to 30 years. He examined the gingivae approximating 165 upper incisors with proximal or cervical caries. These gingivae were found to be normal or only slightly diseased in 54 per cent or a little more than one half of the areas examined and, therefore, King stated that there is no difference in the gingivae surrounding carious and non-carious teeth. He further noted that the frequency of caries in the upper anterior teeth increased with age while there is a tendency for the extent of the gingival lesions to diminish. These observations led him to conclude further that there is no relation between caries and gingivitis.

Savara[5] in an unpublished thesis prepared

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in fulfillment of the requirements for a master of science degree from the University of Illinois, recently has reported the relationship he observed between caries, gingival disease and malocclusion in 2,774 Chicago high school students ranging in age from 14 to 17 years. Under the direction of Drs. Schour and Massler, he applied the P.M.A. method which they have developed for quantitatively assessing the amount of gingivitis. The amount of caries was scored by the D.M.F. index that Klein and Palmer used in their Hagerstown, Md., study.

Correlation between the incidence of dental caries and gingivitis revealed that only in the group with severe gingivitis was the caries incidence high. There was no difference in the caries experience of those classed as having zero, very mild, mild or moderate gingivitis. The conclusion from their overall survey of the group was that no relationship exists between the number of carious teeth or carious tooth surfaces and the number of inflamed gingival units at this age level.

Thus we see the confusion that exists about an association between these two conditions even though here a casual relationship might be expected.

What does the literature reveal about relationship when periodontal breakdown occurs and where suppuration and other end products of soft tissue destruction exist. Many opinions are expressed yet very few articles were found that report a clinical study to check the hypothesis that there is an inverse relationship between caries and periodontal disease.

Broderick[6] has claimed an antagonism between caries and chronic periodontitis. His observations lead him to believe that a state of generalized acidosis is predominate when dental caries is active and that alkalosis provides the background for periodontal disease. He draws from statements by Bibby, Price, Pickerill and Prinz for support in his contention. Others who have made chemical analyses of periodontal subjects have not confirmed Broderick's views. Citron[7] found that the alkalinity of the blood diminished in periodontally diseased individuals and Landgraf[8] made similar observations.

Waiss[9] on the other hand did not agree with either of the above observations and stated that the alkali reserve of the periodontal subjects he studied was well within the normal range. C. F. Boedecker,[10] however, agreed with the views of Broderick.

Waugh[11] has reported that primitive Eskimos were free from caries but had extensive periodontal disease. Barnes,[12] on the other hand, believed from his observations on the Isle of Tristan da Cunha that a rise in incidence of one disease was associated with an increase in the other condition and J. G. Turner[13] stated that in man, caries and periodontal disease can run a parallel course.

Miller and Seidler[14] published a report in 1940 that describes a statistical study on the incidence of dental caries and periodontal disease. It is one of the few in which factual data was collected. One thousand and three individuals were observed and they ranged in age from 11-60. To be included in the study each was required to have a minimum of sixteen teeth. The Boedecker modified caries index was used to compute caries activity and a special classification was developed to measure periodontal destruction. Each mouth was divided into four sections, upper and lower anterior—upper and lower posterior. The condition of the alveolar bone was rated according to the appearance in full mouth roentgenograms. The classification ranged from 0 to 5 for each section. Zero represented no alveolar destruction and five indicated the complete loss of bone to the apices of the teeth. The numbers 1, 2, 3 and 4 represented increasing destruction between the two extremes. The amount of alveolar destruction for each mouth was determined by adding the score given each of the four sections. Thus the count could range from 0 to 20. When the caries and periodontal scores for the 1,003 subjects were compared it was concluded that no correlation exists between caries and periodontoclasia. I interpret this statement to mean that in their

opinion caries and periodontal disease are independent but not necessarily incompatible.

More conflicting and confusing statements could be included in this report but I would only belabor the point that we do not yet have the answer. I believe that one reason why the clinical studies and statements of clinical impressions are varied is that the subjects of study have not been observed over a sufficiently long period of time to draw valid conclusions. Both caries and periodontal disease, as a rule, are slowly progressive. Many of the conclusions are based on one or two observations and single mouth examinations record only the evidence of past pathologic activity. Because the results of the two destructive diseases can be seen at the time an oral inspection is made, it is not necessarily indicative that both processes operated at the same time. We are now in possession of tests which indicate the activity of dental caries at a particular moment and we have tests that appear to do the same for periodontal destruction. If these tests were utilized and their findings correlated with clinical evidence collected periodically from the same individuals over a sufficiently long period to note the clinical progress of the diseases in question, we would be able to arrive at some definite scientific conclusions.

While the clinical picture is confused, certain chemical and bacteriological studies suggest that there may be a fundamental difference in the oral environment which makes the simultaneous activity of these two diseases incompatible.

Berg, Burrill and Fosdick[15] have determined the putrefactive rate of saliva by measuring amino acid, indole and sulfide production. Analyses of saliva were made after one and three hours' incubation. A comparison was made between one hundred individuals classed as normal and 100 showing periodontal disease. It was concluded that saliva from pathological cases putrifies more rapidly than does saliva from normal mouths. Because the correspondence between the roentgenographic, the clinical and the chemical findings was so high the investigators state, "If further studies confirm these results, it may be possible to predict whether a patient is susceptible to periodontal disease and in need of preventive therapy, just as it is now possible to predict with fair accuracy whether a patient is susceptible to dental caries and requires preventive measures."

Dreizen and Spies noted[16] that pellagrins and individuals with long standing nutritional deficiencies had a relative absence of carious lesions particularly of the smooth surface variety but had extensive periodontal involvement. They hint that the observed resistance to caries may result at least in part from an increase in the end products of putrefaction such as indole, skatol, phenol and cresol. They tested exceedingly dilute solutions of these chemicals and various mixtures of them for their ability to stop fermentation in caries active saliva and to inhibit the growth of lactobacilli and they found them to be exceedingly effective.

Berg, Lundquist, Schram, and Fosdick[17] suggest that the glycogen content of the gingivae may have an important relationship between the antagonism which they believe exists between caries and periodontoclasia. They found that chronically irritated gingivae had significantly less glycogen than did normal gingivae. Surgically traumatized gingivae also showed a reduction in glycogen. They postulated that "all those factors which on a clinical basis seem to be causes or contributing factors in chronic periodontitis such as mechanical irritation whether from calculus, trauma, bacterial infection, or some endocrine disturbance, and dietary deficiencies will alter or disturb the glycogen content of the tissues. In the second place, the absence of glycogen, a potential source of sugar, seems to promote protein breakdown and putrefaction." They point out that the various putrefaction complications of untreated diabetes, the failure of Vitamin C deficient tissues to heal, and the alkaline reaction in non-healing tooth sockets may be associated with a decreased glycogen content of the tissue and they also suggest that the terrible periodontal conditions of obvi-

ously malnourished war prisoners might have been due to low tissue glycogen.

Some years ago Hill[18] observed that when saliva collected from individuals with active periodontal disease is inoculated with lactobacilli obtained from a caries active mouth the lactobacilli are destroyed within twenty-four hours despite the addition of glucose to support their proliferation. In caries active saliva there is marked proliferation of lactobacilli during the twenty-four hour incubation period. Studies we have performed indicate that ammonia will form more rapidly in saliva collected from individuals with periodontal disease and that ammonia in concentrations as low as 0.5 mg. per ml. of saliva will inhibit the growth of lactobacilli.[19] In a recent study of one hundred and two individuals in our laboratory, twenty-two were classed as having periodontal disease based on clinical and x-ray findings. Salivary lactobacilli counts in twenty were zero. Two gave rather high lactobacilli counts—both were wearing partial dentures. It might have been that the periodontal destruction was no longer progressive in these two or that the high counts were due to the partial dentures for Bradel and Blayney observed that denture wearers have increased lactobacilli counts. A study of the 102 subjects indicated that those who formed calculus had few or no cavities and suggests that there may be an agency that predisposes to calculus formation and inhibits cavity development. However, this observation needs further study for confirmation and understanding.

CONCLUSION

Chemical and bacteriologic studies indicate a basic fundamental difference between caries active and periodontal destructive oral conditions which leads to the belief that the two conditions cannot be active at the same time. However, clinical observations are controversial and the methods employed have not been sufficiently critical. Clinical studies should be undertaken supplemented by the newer laboratory techniques that determine caries and periodontal susceptibility. The clinical and laboratory findings should be correlated for a large group over a period of at least two years during which the clinical progress of the diseases can be watched. Such studies should determine the compatibility or incompatibility of these two prevalent disorders.

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Editorials

Notes from Dr. Dickson G. Bell

President of the American Academy of Periodontology

THE AMERICAN Academy of Periodontology has as one of its announced objectives the dissemination of knowledge concerning the prevention and treatment of periodontal disease and information in the related fields of science. Membership in the Academy implies an acceptance of this responsibility and an opportunity to serve the profession at large in this capacity. Our Journal of Periodontology is one of the vehicles by which proper information in our special field is presented to the general practitioner.

With this in mind and stimulated by the increased interest in the problems of periodontal disease, we must all accept our responsibilities and work tirelessly to extend the effectiveness of our Journal. Only through propagation of good literature, widely read, will the general practitioner enhance his concept of diseases of the supporting structures.

Every President has objectives that he would like to see achieved during his administration. One of the several I have in mind, probably the most important, is to increase the circulation of the Journal of Periodontology to the members of the dental profession. The Chairman of the Journal Subscriptions Committee has a plan which will be submitted to our membership within the next few months. It is my earnest wish that every member pledge his whole-hearted support toward reaching this goal and co-operate in securing subscriptions whenever possible.

The profession and especially teachers and writers on periodontal subjects look to this Academy for guidance on periodontal nomenclature and classification. The Academy through the efforts of the Nomenclature Committee has an accepted glossary of periodontal terms which is being enlarged each year. The time has now arrived when it is imperative that some agreement be reached on the classification of the various disturbances of the periodontium. It would not necessarily mean that we would be bound to it for all time, as subsequent committees would also have the authority to suggest revisions. It is my hope that the Committee on Classification will reach some basis for agreement that may be presented at our next meeting in Atlantic City.

Your expression of confidence in conferring upon me this outstanding honor —the Presidency of the American Academy of Periodontology is acknowledged with deep appreciation. I feel keenly the trust you have placed in my care and with your co-operation we can accomplish much in the realization of our objectives. I wish too, to solicit your suggestions and recommendations at any time during the ensuing year.

Vincent's Infection by Any Other Name . . . . . .

ONE OF the requirements of a true profession is that it have an exact terminology which is stabilized, well understood and widely used. Periodontia is gradually developing such a terminology. Elsewhere in this issue is published the report of the 1949 Nomenclature Committee of the American Academy of Periodontology, and careful study will show few changes recommended by this Committee from those terms recommended by previous committees.

There will always be some differences of opinion regarding terms, but as they are debated and discussed from year to year there is a gradual unification. Group after group are recognizing the virtue of using standard terms because after all, a term means only what it is defined to mean. It is somewhat absurd to debate minor deviations and spelling of terms when the actual meaning is dependent upon current usage. It can be shown by any student of etymology that terms change from generation to generation and to assume that the meaning of any word is dependent upon the meaning of its Greek or Latin roots is indefensible. For example, the word "anaphylaxis" was coined about 1900 to mean "against protection." The man who invented the term joined the Greek words "phylaxis" (protection) and "ana" (against). The term anaphylaxis is now used for a form of hypersensitiveness, and efforts to make the derivation suit the term lead to absurd explanations. It is pointless to try to explain anaphylaxis by defining the Greek roots that make up the word.

One might criticize many terms that are now being recommended because they are not ideal, but from year to year the terminology will no doubt be "purified" so that eventually an accurate acceptable series of terms for describing periodontal conditions will be available. For instance, we recognize that the use of the term "Vincent's infection" leaves much to be desired. It is generally accepted that diseases should not carry the name of an individual but should be more descriptive; hence, we should use a descriptive term, as "necrotizing gingivitis" or perhaps "necrotizing ulcerative gingivitis." (To one who worships at the shrine of simplicity, however, the latter term seems unduly complex.) One cannot deny that the term "Vincent's infection" is well understood by all dentists and is quite well intrenched in the minds of the public. We should not, however, let common usage prevent us from using more accurate and descriptive terms. While we admit that "Vincent's infection" is an adequate term in some respects, it is our opinion that we should continue to strive for improving our terminology until we can use descriptive terms.

Another common mistake is made in the naming of the organisms involved in "necrotizing gingivitis." The most recently published text in the field of periodontia mentions that the "*Bacillus fusiformis*" is believed to be associated with the disease. When the term "bacillus" is capitalized and italicized, it is then considered a specific generic term, which includes spore-forming bacteria with peritrichal flagella. Members of the genus "*Bacillus*" are in no way related to the bacteria involved in a necrotizing gingivitis. It is permissible to speak of the

"fusiform bacillus" because a bacillus is an elongated rod-shaped microorganism, but the capitalization and italicization of the word "bacillus" places a stamp of specificity upon it. The correct generic term for the organism is *Fusiformis* or *Fusobacterium*. One should speak, then, of a fusiform bacillus (any "cigar-shaped" microorganism) or *Fusiformis dentium* (a "cigar-shaped" microorganism which is anaerobic, stains unevenly, is normally found in the oral cavity of the adult, and is present in increased numbers in necrotizing gingivitis).

If one thinks this is "splitting hairs," it should be pointed out that bacteriologists are accustomed to dealing with minutae. Since it costs no more to be correct, efforts should be made to use accurate, scientific terms.

Physiological Occlusion and Occlusal Trauma

By HUNTER ALLEN, D.D.S., *Birmingham, Ala.*

AMONG the many etiological factors incident to periodontitis, none has received more emphasis than that of occlusal trauma. It, like nutrition and metabolism, has had much written, though little clinical evidence to the effect that a solution has been found. There are cases that respond satisfactorily, yet when others fail under apparently the same conditions, we are convinced that there is much to be learned.

The pattern of all mankind varies and, at times, establishes dental abnormalities such as short roots, large and small crowns, thin alveoli, malposed and malocclude teeth. There is also a variation in soft tissue composition. In such instances inflammation and irritation may be misinterpreted, with emphasis being placed on faulty occlusion rather than some morphological factor. In many cases this could be normal for the pattern. By normal I mean that regardless of the size of an organ, or its position or its contents, if by its function it assists in maintaining a physiological balance and promotes health, it is normal.

The pattern was made at the time of conception; the variations we see later are in accord with inherent factors, or governed by the determination of genetics. There are many ways by which this pattern may be changed, and some of these conditions are being corrected by judicious care. In so doing, thought must be given related parts to be sure that a physiological balance is maintained.

The loss of one or more teeth permits others to drift out of position, breaking the continuity of the arch, disturbing the temporomandibular articulation, with the condyle head shifted distally. The architecture of the alveolar process is jeopardized since the periodontal fibers and bone trabeculae, developed to absorb stimulating forces in a given direction, is being changed. The masticatory stroke, which originally transmitted stimulating stresses to the foundation, can very easily, and frequently does, become one of irritation.

To restore a physiological occlusion, consideration must be given the bio-mechanism in the involved area.

Along with the loss of teeth, changing tooth form and position tend to alter this pattern and will, no doubt, affect the correlated parts, principally the muscles of mastication. By the same token, in the frail alveolus supporting short roots where microdontia appears to predominate along with a seemingly lost verticle dimension, morphological factors are too often disregarded and mechanical procedures adopted with disastrous results.

Brodie (1) states: "It has been shown for example, that if this position in the so-called bite raising operation and the mandible is depressed to a level below that dictated by the muscular balance that controls the bone, the mandible will return to its original resting position within a period of a year even if it has to destroy the alveolar process to do so."

In presence of such changes the roentgenogram will invariably show a coronal rarefaction that also involves the apical of the affected area. This is a typical artificially induced traumatic, or dynamic, irritant in that the masseter and internal pterygoid, the two powerful muscles of closure, are not working in harmony, but for harmony in other parts of the masticatory apparatus. They are constantly contracting, mostly in a vertical direction, parallel with the long axis of the tooth, with forces that greatly exceed physiological limitation.

This action is the effort to restore the temporomandibular articulation to a different arrangement than that recently acquired. The vertical force, which under normal conditions should stimulate and contribute to tooth supporting structure, is now depressing the teeth into the alveolus by continuous pressure of such magnitude that the factors constituting bone recovery are unable to function and irreparable damage is being done.

Injudicious orthodontic shifting and subsequent relapse to near original position often affects foundational tissues detrimentally, in that, what should have been a stimulus becomes an irritant.

Again to quote Brodie: (2) "As far as the specific growth of the face is concerned, it has been shown that the maxilla grows on practically all of its superior and posterior surfaces against relatively fixed cranial bases, so that there results a pushing downward and forward movement of the entire facial mass away from the brain case. The mandible grows similarly in an upward and backward manner. All of this growth is so beautifully integrated that no change in relationship takes place. There is one, independent addition, of course, which does not occur unless teeth are present."

He further states: "The upper jaw may be endowed with a quite different potential than that of the lower jaw, and there are undoubtedly cases where the muscles and bone system are not in harmony. Doubtless, other items could be mentioned in which dysplasia could be demonstrated."

Assuming there is a displacement of the upper anteriors, the removal of the upper first biscupids will permit adjacent anterior teeth to be moved distally with a minimum amount of bone disturbance and relationship with the posterior teeth. There has been little or no functional change of the masticating muscles and the physiological balance can be more satisfactorily obtained.

The question is often asked, "Why then, do some conditions, as mentioned, show a variation in structural changes in both soft and hard tissue?"

One answer is that we are dealing with different individualities. It should be remembered that in building, and in destroying bone, one of the factors is stimulation; just when it becomes favorable for construction or that of destruction is an individual problem. And further, we should know, that success requisites in treating a disease condition are not always bound by text-books, but by the skin, of the individual.

The functional demand required of the alveolus is great, and there are many systemic conditions that interfere with response to the stimuli. Some are inherent, while others are acquired.

Of the inherent, one responsible for many defects in bone, which manifestations are not seen until later in life, is due to development of tissue arising from the mesenchyme. If a hypoplasia of this portion of the embryonic mesoderm has existed, connective tissue

1. Brodie, A. G., *J.A.C.D.* **15**:161, 1948.
2. Brodie, A. G., *J.A.C.D.* **15**:161, 1948.

is affected. Individuals with such arrangements are very likely to suffer some bone afflictions such as Rickets, Osteomalacia, Osteo-fibrosa, and many other dysplasias that tend to slow up ossification, making the bone more susceptible and intolerable to stresses.

The acquired, such as deficiencies of minerals, hydrocarbonates, and their derivatives, the vitamins, the endocrines; diseases that interfere with assimilation of these food elements, and make tissues susceptible, not only to stresses, but other detrimental influences.

When such a dystrophic condition exists and a physiological balance is desired, reshaping of the teeth in the involved area may be indicated. In doing so, it should be remembered that this, as a rule, requires grinding, which does one of two things, either the stress is increased or decreased.

If there is an increase in stress, there will likely be further destruction. If there is a decrease in stress, there may be a lack of stimulation.

Before any attempt is made in correction of a weakened supporting structure, there should be a reduction of the "working table" of the affected tooth, to take care of the lateral force along with accentuation of the fossas. If there are only a few areas that show strain, these should receive attention, rather than the entire mouth. If one had thirty-two or twenty-five persons working for him and two or four became ill, should all be given the same remedy? Of course, if an epidemic exists, then there is a different story.

To re-establish a physiological occlusion, one should know the precise requirement for normal tooth form and function if an abnormal supporting structure exists.

Present Status of Periodontia as a Specialty

By ARTHUR H. MERRITT, D.D.S., M.S., SC.D., F.A.A.P., *New York, N. Y.*

PERIODONTIA may be said to have had its birth as a specialty in dental practice with the organization of the American Academy of Periodontology in 1914. While diseases of the periodontium are as old as recorded history and some attention had been given them, little progress had been made in the way of an organized attempt toward their prevention or treatment. Fauchard in 1746, Hunter in 1778, Kunstmann in 1772, Robicsek in 1894 in Europe, and Riggs, Younger and Smith in this country, each made, in his way, some contribution to periodontia and were usually referred to as "pyorrhea specialists."

However, it was not until the organization of the American Academy of Periodontology that a systematic effort was made to give to periodontia its logical place in the practice of dentistry.

Beginning as pioneers and with no precedent to guide them, five members of the dental profession came together on February 21, 1914 to consider the formation of an organization for the promotion of periodontia. After prolonged discussion, they adjourned to meet in Cleveland on May 23 of the same year, at which time the American Academy of Periodontology was brought into being.

Among the first of its many activities was that of education, to include both the public and the dental profession. To this end, educational bulletins for the public, covering every phase of dental practice were prepared and given wide publicity. To aid the general practitioner, a section of periodontia in the American Dental Association was formed, meeting in 1919 with the section of orthodontia and becoming a section by itself ten years later. In 1929 THE JOURNAL OF PERIODONTOLOGY was founded for the further promotion of periodontia.

I

Such, briefly, is the background of periodontia as a specialty. At present it may be

said that many of the activities of the past, expanded and made more effective, are in operation, plus the advances born of experience. The section of periodontia in the twenty years of its existence, has become one of the best attended sections in the American Dental Association. Increased interest in periodontia is evident on every hand. A larger number of general practitioners are becoming aware of its importance and are advising their patients accordingly. Every year adds to the number of those seeking certification. At the same time, those engaged in the practice of periodontia are becoming more resourceful. Less teeth are being condemned to extraction and more attention is being given to prevention. THE JOURNAL OF PERIODONTOLOGY has been increased to a quarterly with subscribers in a dozen or more foreign countries.

All this when added up, indicates that substantial progress has been and is being made with promise of further advance in the future. This is encouraging.

II

The goal, however, has not been reached. First and foremost in the continued advancement of periodontia, is *research*. Until more is known regarding the cause and effect of periodontal diseases and their relation to the body as a whole, complete success in their prevention and treatment will not be possible.

No student of the subject can have failed to be impressed by the fact that there are cases presenting for treatment, sometimes referred to as the systemic type, which cannot be explained by local conditions. What are the factors responsible for the condition seen in these cases? Is it dietetic, nutritional or heredity? At present, no one knows the answer.

The influence of occlusion on the periodontium is still unknown notwithstanding all that has been said and written on the subject. Is it a cause or an effect, or both? Again, no one knows the complete answer.

Research into the hidden causes of these and other factors will doubtless reveal influences affecting the periodontal tissues at present wholly unsuspected. Increasing knowledge has a way of pushing back the barriers of ignorance, at the same time widening one's vision to see, as it were, the invisible. Research must, therefore, be regarded as essential to continued progress in periodontology.

III

While this is true, it is also true that the knowledge already available in the field of periodontology, is not being made use of as widely as it should be. About this there can be no doubt. The periodontist is made painfully aware of this every day in his office. With no desire to be critical, it must be said that the responsibility for this situation is largely that of the dental schools. It would appear that they are to some extent at least, still under the influence of the mechanical concept of dentistry. The emphasis is too often being placed on mechanical procedures. That these are important and make up a large part of daily practice, no one will deny, but they are neither the beginning nor end of dental practice, nor are they the most important part. Much of the mechanics of dental practice could be eliminated were more attention being given to the prevention and treatment of periodontal diseases.

What is needed in the dental school is a department of periodontology presided over by one who is not only a good teacher but an experienced clinician, and one able to inspire in the mind of the student the fundamental importance of periodontia in the successful practice of dentistry. Too often is the student graduated with little or no conception of the meaning of periodontia and with less skill in its practice. This is a situation which must be referred to in any appraisal of the present status of periodontia. It must be said, however, that the fault is not entirely that of the dental schools. Periodontia as a specialty is still so young that competent teachers in the field are not always available. This is a situation which only time can wholly remedy though improvement is possible under present conditions.

IV

Another weak link in the present status of periodontia is the general practitioner. Again with no desire to be critical it must be said that too often the general practitioner fails in the periodontal care of his patients. This can be explained in part by inadequate undergraduate training. It was not so long ago that little or no instruction in periodontia was being given in our dental schools. This, however, does not absolve the family dentist of the present from failing in his care of his patients in this respect. If he has kept in touch with the progress that is being made in dentistry through study and attendance at meetings, he will have no difficulty in recognizing the periodontal needs of his patients. A careful examination of the mouth *as a whole*, when done periodically, will reveal the presence of periodontal diseases should any be present. If the general practitioner should feel that he is not competent to give the needed treatment, he will have discharged his obligation to his patients by referring them to one whom he knows to be expert in this field.

V

In conclusion it may be said that the present status of periodontia as a specialty is one in which substantial progress has been made; that never in the history of dentistry was the knowledge of periodontia so widespread or success in treatment at so high a level; that research, better undergraduate instruction and a clearer understanding of the importance of periodontia in general practice, are among the progressive changes that must take place before periodontia as a specialty will be able to meet the needs of the public.

Report from the 1949 Nomenclature Committee
of the American Academy of Periodontology

No subject can be considered a true science until it has a standardized nomenclature which is generally accepted and widely used. One of the important functions of the American Academy of Periodontology, therefore, is to continue to modify and clarify the terms used in periodontology, and to add to and delete from an accepted list of terms as soon as our knowledge and experience justifies a change.

The 1949 Nomenclature Committee is somewhat pessimistic regarding the possibility of this and any similar report receiving immediate and enthusiastic acceptance. Most periodontists have used their own terms for so long that any suggested change is resisted and resented. Furthermore, this and any similar report represents the personal opinions of its committee members, and membership on the committee merely allows a new group of men to have their particular system of terminology accepted. For this reason we believe the Nomenclature Committee membership should serve for at least three years.

The 1949 committee noted that there is a tendency on the part of many authors to coin unnecessary new terms or to use terms which are unduly complex. This committee believes such a trend is undesirable and, hence, recommends that the members of the American Academy of Periodontology discourage the use of such words as ginginoplasty and odontexesis.

Although presenting the system of terminology acceptable to everyone is impossible, it is essential that this committee continue its activity to help bring order and uniformity into the field of periodontia. This committee, therefore, wishes to submit the following list of terms:

1. *Alveolar bone:* The alveolar processes of the maxillae and the mandible. Within the alveolar bone an arbitrary division can be made between the "cribriform plate" and the "alveolar supporting bone." The cribriform plate may be defined as the thin perforated bony plate to which the fibers of the periodontal membrane are attached and

through which the vessels and nerves enter and leave the periodontal membrane. In radiography this bony plate is called "lamina dura" as it appears more radiopaque than the surrounding spongy bone, but the term "cribriform plate" is to be preferred. The alveolar bone consists of the compact cribriform plates and of the spongy or travebular bone of the alveolar processes.

2. *Alveolus, dental:* The socket in the alveolar process in which the root of the tooth is ordinarily held by the fibers of the periodontal membrane, and which is normally lined by the cribriform plate and surrounded by alveolar supporting bone.

3. *Bruxism:* Gnashing or grinding of the teeth in other than normal function. This grinding of the teeth is an unconscious habit and is often limited to the sleeping period, but may occur at times of mental or physical concentration or strain. Bruxism may develop into a neurotic symptom, "bruxomania."

4. *Calculus:* see—Dental calculus.

5. *Dental Calculus:* (synonym—tartar) Concretions deposited on the teeth consisting of inorganic salts deposited in an organic matrix. One can classify dental calculus roughly into supra-gingival calculus, and sub-gingival calculus.

6. *Epulis:* The term "epulis," denoting a tumor on the gingiva, is a non-specific term, and should be discontinued. The form is loosely used for tumors of varying cell types.

7. *Gingiva:* That part of the oral mucous membrane that covers the alveolar processes of the jaws and surrounds the necks of the teeth.

8. *Gingival hyperplasia:* Overgrowth of the gingival tissues. There is some misunderstanding as to the correct use of the terms hyperplasia and hypertrophy. A sharp and definite difference does not always exist. Hyperplasia is defined as an overgrowth of tissue due to an increase in the number of its elements. Hypertrophy, on the other hand, is an increase of an organ due to increase of the size of its elements, and serves a useful purpose. An example of a true hypertrophy is the enlargement of the heart due to overwork, or enlargement of muscles due to increased activity. Each cell becomes longer and thicker. Overgrowth of the gingiva, however, is a hyperplasia (increased number of cells).

9. *Gingival pocket:* The gingival pocket may be defined as a pathologically deepened sulcus.

10. *Gingival recession:* Progressive exposure of the root surface by apical shift of the gingiva, following resorption of the alveolar crest and loss of the corresponding part of the periodontal membrane. (See—periodontal atrophy.)

11. *Gingival sulcus:* The shallow groove around a tooth, bounded on one side by the surface of the tooth and on the other by the epithelium lining the free margin of the gingiva. This term designates the "normal" condition as distinguished from the gingival and periodontal *pocket*.

12. *Gingivitis:* Inflammation of the gingiva. The condition may be further defined and described by such adjectives as acute, chronic, purulent, ulcerative, hyperplastic, scorbutic, etc.

13. *Intrabony pocket:* This term is used to describe a pathological pocket whose base is markedly apical to the alveolar bone crest for the adjoining tooth. The term is not a good one because it implies that the pocket is "within" the bone. While all members of the Committee agreed that the term was misleading, no acceptable substitute was suggested, although "interbony pocket" (pocket alongside of bone) would be better than the present term. "Intrabony pocket" should be deleted as soon as a more satisfactory one is found.

14. *Oral Mucosa:* (or oral mucous membrane) is the lining of the oral cavity. It may be divided into: *masticatory mucosa*—Epithelium hornified, immovably attached; gingiva, hard palate; *lining mucosa*—Epithelium nonhornified: loosely attached, firmly attached; *specialized mucosa:* Back of tongue.

15. *Periodontal abscess:* Acute or subacute purulent exacerbation of a chronic periodontitis, associated with deep periodontal or gingival pockets.

16. *Periodontal atrophy:* Progressive resorption of alveolar crest with loss of the corresponding part of the periodontal membrane, recession of gingiva, and exposure of root surface.

17. *Periodontal diseases:* Collective term designating any disease process which involves the periodontium.

18. *Periodontal membrane:* The fibrous tissue that connects the cementum of the tooth to the surrounding structures.

19. *Periodontal pocket:* A periodontal pocket may be defined as a gingival sulcus pathologically deepened by periodontal disease. It is bordered on one side by the tooth and on the other by the gingival tissues. Its base is limited by the epithelial attachment, which is usually not intact in this area. The chief features of the periodontal pocket are the ulceration of the epithelium lining the pocket and the inflammation of the gingival corium. The "pocket" is usually filled with debris and calculus.

20. *Periodontal traumatism:* Injury of periodontium due to excessive occlusal operative, accidental or orthodontic stress (see—traumatism).

21. *Periodontist:* One who has acquired considerable knowledge of periodontology and is skilled in the treatment of periodontal diseases.

22. *Periodontia:* The branch of dentistry that deals with the science of periodontology and the treatment of periodontal diseases. Periodontics is an acceptable synonym.

23. *Periodontitis:* Inflammatory involvement of the periodontium. *Periodontitis simplex*—sequela of gingivitis. *Periodontitis complex*—periodontitis secondary to periodontosis. The condition may be further defined and described by such adjectives as acute, chronic, ulcerative, purulent (suppurative), etc.

24. *Periodontium:* The investing and supporting tissues of the tooth, namely the periodontal membrane, the gingiva, cementum and the alveolar bone. Although the cementum is part of the tooth, it is one of the supporting tissues because it holds one end of the periodontal membrane.

25. *Periodontology:* The science and study of the periodontium and periodontal diseases.

26. *Periodontosis:* A general term to include a degenerative non-inflammatory destruction of the periodontium originating in one or more of the periodontal structures, characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease. It is possible to subdivide cases of periodontosis into several sub-divisions.

27. *Pocket:* See—gingival pocket, periodontal pocket, intrabony pocket.

28. *Sulcular (Sulcate) epithelium:* The thin epithelial lining of a gingival sulcus.

29. *Traumatism:* Morbid condition of tissue caused by mechanical trauma (pressure necrosis and its consequences: thrombosis, hemorrhage, resorption of bone and cementum).

30. *Vincent's infection:* It is recommended that this designation of a certain type of gingival inflammation be discarded. The term was used to designate an acute ulcerative gingival inflammation. Properly one should speak of an "acute, sub-acute or chronic necrotizing gingivitis." The term "Vincent's angina" should be discarded.

The following terms are suggested as a glossary for the technics used in periodontal therapy:

Periodontal Prophylaxis: Technic for prevention of periodontal disease, which includes cleansing and polishing of teeth, elimination of irritating margins of fillings, correction of occlusion, etc.

Sub-gingival curettage, gingivectomy, flap operation, occlusal equilibration, occlusal adjustment or correction, functional adjust-

ment or correction, prophylactic scaler, prophylactic curette.

SUMMARY

The 1949 Committee on Nomenclature makes the following recommendations:

1. That in the future the Nomenclature Committee be appointed for three years.

2. That the terms submitted in this report be accepted officially for the American Academy of Periodontology.

Since the first recommendation may require an amendment to the Academy's by-laws, we wish to move that this report be submitted to the Executive Council for further consideration.

HARRY LYONS
DONALD M. KERR
MAYNARD K. HINE, *Chairman*

Book Reviews

Periodontia

By HENRY M. GOLDMAN, D.M.D.

A new edition of Dr. Henry Goldman's "Textbook on Periodontia" is now available and deserves a good reception. Dr. Goldman, who is Chief of Stomatology and head of the Dental Department at Beth Israel Hospital in Boston, periodontist of the Massachusetts General Hospital in Boston, and consultant of the Army Institute of Pathology in Washington, D. C., is well versed in practical and theoretical considerations of periodontia and so can write with authority.

This second edition is over two hundred pages larger than the first edition, which was published in 1942. While expressing the same general points of view, Dr. Goldman has improved the book by rearrangement of text material. The first two hundred pages include a discussion of the normal histology of the periodontium, then a consideration of the pathologic changes noted in the periodontium. This is followed by a detailed summary of the etiology of periodontal diseases, and a discussion of experimental pathology related to the tooth attachment apparatus.

The author then presents the problems of examination, diagnosis and treatment of periodontal diseases. New chapters added include one on "necrotizing ulcerative gingivitis," one on medicinal aids in periodontal therapy, and one on diseases of the oral cavity. Good lists of references are added to each chapter. While most of the 488 illustrations are excellent, several of the clinical photographs are not reproduced too well. In general, however, the pictures are valuable in supplementing the text. The general organization of this edition is improved and students will find it easy and profitable to read.

Dr. Goldman quotes liberally from dental literature to supplement his original contributions. As a result, the book is a good summary of current status of opinion in periodontia. It is worthwhile to note that the author has used the nomenclature which is accepted by the American Academy of Periodontology. The book should be in every dental library and will be valuable to the serious student of periodontia.

It is published by the C. V. Mosby Company of St. Louis and sells for $12.50.

Cunningham's Manual of Practical Anatomy. Revised and edited by J. C. Brash.

This new edition of the well established "Cunningham's Anatomy" is the eleventh of a series since the first "Disector's Guide" was published by Dr. Cunningham in 1879. There are three volumes in this edition, Volume I dealing with the extremities, Volume II with the head and the brain, and Volume III with the thorax and abdomen. Included is a glossary comparing the Birmingham revision and the Basle Nomina Anatomica.

The books are clearly written and amply illustrated. Included are many radiographs which are particularly helpful to the student in correlating the anatomic structure as seen in dissection with that seen by radiography. The volumes are small and compact.

Cunningham's Manual is published by the Oxford University Press, 114 Fifth Avenue, New York 11, New York and costs $12.75 for the three volumes, or $4.25 each.

THE PERIODONTAL LESION

(Continued from page 18)

which manifest themselves in the gingiva as well as other body tissues.

It is important at this time to clarify the reaction of bone in the development of the periodontal lesion. It has been previously pointed out in the discussion on inflammation that under the influence of the inflammatory reaction, the mineral content of the the bone matrix becomes soluble. When the greater part of the inorganic matter of the alveolar crest goes into solution, only the matrix remains. The organic matrix may undergo degenerative changes nearest the site of the reaction, but it should be emphasized that at this point the bone itself is not infected but is merely reacting to the local acidity built up by the inflammatory process. If the irritants are eliminated, this reaction is reversible, and a remineralization of the matrix will occur. Should this partially demineralized matrix be surgically removed or interfered with by operative procedures, it may be destroyed, and no bony repair can take place beyond the height of the operated crest. It is of great importance to maintain the organic matrix if a maximum amount of repair is to be achieved.

ON THE THEORY OF TREATMENT OF PERIODONTAL DISEASE

Treatment planning in periodontal disease demands an awareness of the many etiological factors that might be involved. It must not only be recognized that systemic complications, if any are present, must be diagnosed and, if possible, brought under control, but that all other known factors, local, anatomical and functional, must be given consideration. In addition, a definite effort should be made in making oneself aware of the possible daily emotional trials besetting the patient. The purpose of treatment is to arrest the advance of the disease and *to maintain the teeth and the periodontium in functional repair* for as long as possible.

Systemic factors aside, control of the local periodontal lesion should be carried out by a method which will eliminate the inflammatory reaction, reduce the depth of the pocket, and conserve the remaining supporting structures of the teeth. A great deal of argument has been brought forth as to the best type of clinical treatment. In many text books and in any number of papers the chief consideration seems to be that of treatment or systems of operative procedures, while the basic and fundamental reactions of tissue are only touched upon in a most cursory way. If the dentist is to treat periodontal disturbances in any way other than by empiricism, he must necessarily be concerned with biological reactions of disease, especially those which are concerned with tissue repair. If he is to prescribe drugs rationally, it is most important that he be familiar with their pharmacological action on tissues.

The objective in the treatment of the periodontal lesion seems to be generally accepted. The majority of dentists agree that there should be a reduction of the depth of the pocket, whether by repair from within the pocket or by surgical removal of the gingival tissues. Some feel that one should strive to reduce the pocket to one or two millimeters in depth, while others are of the opinion that a pocket of greater depth is permissible as long as the tissue remains in a healthy state. It is admitted by all that the desired result would be complete eradication of the periodontal pocket.

While it is not always possible to entirely eliminate the pocket without extracting the tooth, it is often possible, in closely supervised periodontic cases, to control the progress of the lesion—even though the pocket remains of some depth—by the removal of the irritating factors.

In general the local lesion has its inception in the gingival sulcus. The sulcus, even when of little depth, effects a potential area for the accumulation of foreign bodies such as calculus and microorganisms with their toxic products. The accumulation of foreign substances in this area constitutes the irritating factor of the disease. The irritant produces an inflammatory reaction in the subepithelial

tissues. If it is persistent and progressive in character, the initial inflammatory change will ultimately lead to undesirable tissue changes. In recapitulation from previously stated paragraphs, these changes, brought about biochemically, consist of the following: a degeneration in the epithelium, a breaking down of the gingival periodontal fibers followed by a marginal rarefaction of the alveolar crest, a deepening of the gingival sulcus, pocket formation and suppuration from the area, and eventual extension and destruction along the root, resulting in a pocket of several millimeters in depth.

If, however, the irritant is removed before the lesion has progressed to a certain point, the affected part will undergo repair, the process of which will be discussed later in the paper.

PREPARATION OF THE ORAL CAVITY

The first consideration in treating the lesion locally is the preparation of the oral cavity prior to operative procedures. Gingival tissues which are metabolically sick are more susceptible to bacterial action; hence an infectious process can be superimposed upon a tissue whose resistance is subnormal. Thus prevention of bacterial invasion becomes a preoperative measure. A considerable amount of clinical investigation has been done on the use of antibiotics to reduce the oral flora prior to operative procedures. A method which seems to yield good results is the use of penicillin in the form of troches which allows for a slow evolving of the drug for varying periods of time.[25] Another use of penicillin which seems to have merit is the injection of penicillin in a light oil base or aqueously directly into the pocket. These methods used either singly or in combination have met with a high degree of success when it was desired to reduce the bacterial flora in the mouth before operative procedures were undertaken. The possibility of producing a transient bacteremia in unusual incidences is greatly reduced when such preoperative measures are utilized.

The use of oxygen in the treatment of periodontal disease was introduced about 1913.[26] A specially medicated molecular oxygen from a storage tank was carried through a tube to the gingival tissues. The gas penetrated the gingival tissues, and it was assumed that a hyperemia was produced which in some way promoted circulation. At different times this method of oxygen application has been successfully used, and many clinicians have found it a valuable adjunct in the treatment of periodontal disease.[27, 28, 18]

Whereas in the past chemical agents have been largely used in the cauterization of tissue and the destruction of bacteria, we are now approaching an era in which chemotherapeutic agents are utilized as an adjunct in promoting cell metabolism. These agents which are constituents in the normal cellular oxidative process, such as cytochrome C,[29] are now being utilized in combating local anoxia. Although these drugs have not been proven of benefit in the treatment of periodontal disease, they do offer new avenues of approach which may prove fruitful to the practitioner.

The pursuit of an attempt to promote cellular oxidation in diseased tissues has led to the development of a compound which yields a more readily available supply of oxygen to tissues than does molecular oxygen. This compound consists basically of urea peroxide. Clinical experiments for four years, employing the drug, have continually demonstrated that there is a definite reduction in cyanosis following its use. The experiments also demonstrated that periodontal tissues which received oxygen therapy showed a cellularity closer to that of normal tissue and less tendency toward scar formation.[18]

OPERATIVE PROCEDURES

The first operative procedure to be employed following the preparation of the mouth should be concerned with the removal of the primary irritant. The correction of improperly contoured restorations should be instituted first in the area to be operated. The removal of calculus should be done by careful curettage—adhering to the principles of periodontal instrumentation.[30]

Curettage encompasses not only the re-

moval of calculus, but also careful preparation of the cemental surface of the root, the breaking up of the epithelial lining of the pocket and the establishment of a blood clot. If a functional repair is to be achieved, along with a reduction in pocket depth, the establishment of the blood clot becomes the most important consideration. A brief discussion of the blood clot in the healing processes which occur following the fracture of long bones readily lends itself to emphasizing the necessity for the establishment and maintenance of the clot in the repair of the periodontal lesion. It will be necessary, however, that the preliminary stages which lead to either repair or pathology be again briefly developed to the point with which we are now concerned.

Immediately following the fracture of a long bone a blood clot forms in the fractured space, and the area, encircled by a fibrin blockade, becomes the center of a more or less isolated chemical factory. The tissues of this injured area now must respire anaerobically, and, as a result, lactic acid begins to form in place of the end products of normal cell oxidation. A dissolution of mineral salts from the ends of the fracture results from the local acidosis. This can be demonstrated roentgenographically as a translucency in the fracture area.

At the onset of healing, the blood clot forms the basis for organization and the formation of a new bone matrix in which the bone salts are to be deposited. The processes by which bone or other mineralized tissues undergo repair are precisely the same. The difference in repair of bone, cementum and other tissues of mesoblastic origin lies in the local source of the mineral salts. Anything which tends to disrupt the organization of the clot will inhibit or prevent bone repair. The clinical application would seem to be that in treating a fractured bone, an endeavor should be made as soon as possible to cause a blood clot to form at the site where new bone should grow. After the clot is formed, anything that would interfere with its organization into adult tissue should be avoided—in other words, the sanctity of the blood clot must be respected.

These observations on bone repair can be applied in toto to the processes that obtain in the periodontal lesion where there has been an involvement of bone and cementum. The type of pathological pocket which extends below the height of the alveolar crest should be considered as having a potential for repair to the height of the existing demineralized alveolar crest if a blood clot can be established. An osseous repair beyond this point is unlikely, since with the loss of matrix there is no mechanism which will bring about a regeneration of bone to a normal contour in the area. In order to have a repair of the periodontal fibers, i.e., an attachment with the root surface, new cementum must be formed on the root, and the tooth socket must develop a cortical plate of bone. In order to obtain this objective, surgical intervention in the form of removal of the irritant, curettage of the cementum surface and epithelial lining of the pocket, and the production and retention of a blood clot are necessary.

With the establishment of the blood clot in the pocket, the process of healing parallels that which was seen in the repair of a fractured long bone. The lowered pH in the operated area rarefies the alveolar bone approximating the pocket and the surface of the cementum adjacent to the lesion. It is in this way the operator has established a biochemical lesion which, in the end, produces a tissue repair. As an organization of the clot takes place, a primitive syncytium is formed. Normal metabolic processes come into play as maturation progresses. Fibroblasts, endothelial sprouts and other connective tissue elements from the soft-tissue side of the pocket replace the syncytium. The elementary fibroblasts on the bone side undergo a specialization to form osteoblasts, while those approximating the cementum organize to form cementoblasts. A vascular continuity is established, tissue specialization progresses, and pseudo-periodontal fibers are formed in the area undergoing repair.

The formation and maintenance of a blood clot and the organization of a syncytium are necessary parts of the mechanism if any

repair of the pocket is to be accomplished. The embryonic tissue which develops as the result of the blood clot has the potential for replacing the injured part with a similar tissue; thus the part approximating the cementum will form a cementum matrix, the part near the bone will organize into a bone matrix and both tissues will undergo a mineralization. The central area develops as connective tissue and forms the fibrous attachment. The repair of all wounds involves the same basic tissue changes. These changes vary only with type, location, the extent of the wound and destruction of tissue.

A study of a histological report where trauma produced a root fracture which subsequently underwent repair,[31] indicates that a repair under these conditions is not unlike that which occurs in the periodontal lesion which has been surgically treated by curettage. In this instance of root fracture the fragments either had moved apart or the fracture surfaces had undergone considerable resorption. During the repair process the root ends became covered with cementum, while the intervening space contained connective tissue which corresponded to the periodontal membrane of the root surface.

There is also a report in the literature which demonstrates that following the surgical removal of portions of alveolar bone, periodontal membrane, cementum and dentin from experimental animals, a repair of all three tissues takes place, providing a blood clot is maintained.[32] The above illustrations of repaired periodontal tissues have been introduced in an effort to throw some additional light on the mechanisms which obtain in the repair of the periodontal pocket.

There are cases which might necessitate surgical intervention (gingivectomy) in order to eliminate the pocket. These usually constitute pockets that are bounded by soft tissues not supported laterally by alveolar bone, otherwise referred to as the horizontal type of resorption. In this type of pocket there is no means by which bone can be rebuilt; therefore, a repair in the sense of a cementum-fibrous-bone reattachment cannot be achieved. A pocket of this kind may permit the accumulation of debris, and it is not always possible to maintain the area free from irritation by routine mouth care.

Another type of pocket which may not respond to conservative treatment and often requires the removal of bone as well as soft tissue is the pocket which forms in the bifurcation and trifurcation of molars. In this case it is necessary to have drainage to prevent a recurrence of the inflammatory process. There is no rational justification, however, in removing an alveolar crest on the assumption that a crest is sharp and irregular and thus constitutes a local source of irritation or infection. The injudicious removal of suspected tissue may result in the destruction of a demineralized bone matrix (not apparent in the x-ray) which might otherwise have had a potential for repair.[7] Such a demineralized area is the result of a local disturbance in metabolism and constitutes a biochemical lesion, the reactions therein capable of being reversed. The area will remineralize to the extent of the existing bone matrix providing the irritating factors are removed. Should this area be operated on and the rarefied bone removed, no repair can be expected beyond the height of the operated crest. Consequently, a considerable amount of bone that would otherwise repair is unjustifiably destroyed.

It would seem, from the foregoing discussion, that prior to a surgical removal of the alveolar crest, an attempt should be made to bring the disease under control by more conservative methods. Only when the removal of the irritant and the establishment of the blood clot have failed to bring about a repair of the pocket, should more extensive operative procedures be employed.

It has been previously emphasized that the development and maintenance of a hematoma in bone repair is a most important consideration and anything that would tend to disturb the clot should be avoided. One of the factors most often connected with the loss of the clot in the healing of bone is too much movement at the site of fracture. It has been shown by histological studies of fractures that on the second day a clot lies

at the end of the bone; on the seventh day the site of fracture is seen to have fibrous tissue elements growing in from the traumatized area; on the thirteenth day some cells retain their fibrous tissue form while others have differentiated into osteoblasts, and bone salts can be determined in the extracellular matrix spaces.

From the above data it seems logical to assume that the second most important consideration is the fixation of the movable parts to permit the organization of the clot. Likewise, in the treatment of the periodontal lesion, the maintenance of the blood clot is most important if a maximum of osseous repair is to be achieved; the teeth and tissue of the periodontium must be immoblized for a period of thirteen days or longer, for movement of the teeth in question would tend to mechanically destroy the clot. Hence, in order to promote a better healing in cases where the teeth are mobile, an immobilization of teeth as well as the soft tissues should be given consideration.

SUMMARY

1. In order to put forward a clear picture of events leading to periodontal disease the influence by systemic factors has only briefly been acknowledged in preference to the more stereotyped sequence which is manifested in the development of the local periodontal lesion.

2. The periodontium presents a tissue relationship which makes it an ideal area for the study of tissue reaction to injury.

3. Inflammation is the manifestation of cellular injury by an irritant, and can be demonstrated as being initiated biochemically.

4. Of several factors, the clotting mechanism and cellular metabolism of *glucose* must be especially considered in any tissue injury.

5. Inflammation is the pivotal reaction in tissue response to injury.

6. The development of the periodontal lesion is inaugurated as is any surface lesion and follows the same pattern of progress.

7. The establishment of the periodontal pocket is a result of tissue breakdown, and for the optimum repair (considering all the factors developed in the presentation of this paper) the following operative procedures must be undertaken:

(a) The elimination of the bacterial irritant (medication).

(b) The oxygenation of tissue in order to promote better cell oxidative processes.

(c) The removal of the primary irritant, calculus, (Scaling and curettage).

(d) The scarification of the pocket epithelium and establishment and maintenance of the blood clot.

(e) The splinting of soft tissues and teeth to permit organization of the clot and tissue repair.

(f) Surgery (gingivectomy) if repair cannot be achieved by the above methods.

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Report on the 11th International Congress of the ARPA

The 11th Congress of the International Association for Research upon Paradentopathies (ARPA) was held at the University city of Groningen (Holland) from June 28 to July 2, 1949. Twenty-nine papers on the three themes "Nomenclature and Classification," "Therapy," "Orthodontics and Paradental Disease" were presented by an international assemblage of speakers.

The International ARPA was formed after the Congress of the F.D.I. (International Dental Federation) in 1931 to establish an international forum for the study and discussion of all the problems concerning the paradentopathies. Since 1931 thirty national sections have been established in Europe, South America, Canada and Australia. The Association headquarters are located in Geneve. Dr. R. Jaccard is President. A journal, "Paradentologie," edited every 2 months by Prof. A. J. Held of the University of Geneve, provides for an active exchange of ideas and literature in the three languages, French, English and German.

The September 1949 issue of "Paradentologie" contains a review of all the papers presented at Groningen. Prof. Hulin (Paris), Dr. Orban (Colorado Springs), Prof. Palazzi (Milan), Prof. Held (Geneve), Prof. Ayollon (Peru), Dr. Beyeler (Berne), et al. contributed to the first theme on terminology and gave their own classification. As to the terminology, there are still differences in opinion between the views expressed by the Congress and those held by the Academy of Periodontology, but according to the resolution there will be made a great effort toward a reciprocal agreement.

The second theme comprised 10 papers, among others a study by Dr. R. Jaccard (Geneve) of the part played by heredity and predisposition and a report by Dr. Baume (San Francisco) on Paradentosis and Growth Hormone with reference to the effect of the purified pituitary growth hormone upon the osseous system. Dr. Groen and Dr. Duyvenz (Amsterdam) reported upon the incidence of paradentosis atrophicans in 22 patients with osteoporosis vertebralis and osteoporosis generalisata, and Dr. Chaput (Paris) on "The role of the neuro-vegetative system upon paradental disease." Dr. Kallay (Zagreb) presented a new therapeutical orientation of the paradentopathies by means of 20 intramuscular injections of a placentary extract and electrophoretic introduction of a 1.5% solution of $CaCl_2$ and 1% $SrCl_2$ at the positive electrode and ½% $NaHPO_4$ at the negative electrode (7 m. Amp., 35 volts, 10 times 20 mins.) intragingivally. Dr. Sandoz (Basle) gave a report on functional substances in paradentosis with special reference to the vitamin E, which proves to be of a therapeutical influence and in experimental deficiency a causative factor in paradentosis.

The third theme "Orthodontics and Paradentosis" was discussed by Dr. Muller (Paris), Prof. Parma and Wachsmann (Prague), Dr. Mühlemann (Zurich), Dr. Dujzings (Utrecht) et al.

In a panel discussion the following resolutions were accepted:

1. That the studies on the subject of nomenclature and classification shall be continued in view of the next international meeting of the F.D.I. in London in 1952 and *if possible with the cooperation of the American Academy of Periodontology.*

2. That the terms "Parodontium," "Parodontosis," "Parodontopathies" and other derivatives of the same root in making allowance to the etymological aspect can be used in the same way as "paradentium," "paradentosis," etc.

3. That the studies of the relation between paradentopathies and systemic conditions, as well as between functional disorders, may be encouraged and carried on.

4. That it would be desirable to discuss the common problems of the fields of orthodontics and periodontics during a combined meeting of the European Orthodontic Society and ARPA.

5. That the reports on the treatment of paradentopathies presented at the next meeting of the ARPA Internationale in Madrid, 1951, may be considered largely from the sociological aspect.

6. That paradentosis constitutes a disease sociologically seen also as important as the diseases of the endodontium and that therefore its study and therapeutic approach should cover an adequate place in the plan at Colleges of Dentistry.

L. J. Baume

Announcements of Postgraduate and Graduate Courses

New York University College of Dentistry offers over 60 postgraduate courses in addition to special graduate training. Postgraduate instruction ranges from short refresher courses to complete preparation toward certification in the various subjects.

One and two year postgraduate courses in Periodontia leading to a Certificate of Proficiency in Periodontia can be arranged for specially qualified students whose undergraduate records are outstanding. For information apply Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, New York.

The First Annual Berkshire Conference in Periodontology and Oral Pathology

Tufts College Dental School Postgraduate Division will conduct an advanced conference in Periodontology and Oral Pathology under the direction of Dr. Irving Glickman and staff, at Eastover, a resort hotel in Lenox, Massachusetts, June 13 to 17, 1950. The conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities, with afternoons open for recreation.

The conference faculty includes Mark Aisner, M.D., Assistant Professor of Medicine, Tufts College Medical School, Edwin B. Astwood, M.D., Ph.D., Endocrinologist, New England Medical Center, Lt.-Col. Joseph L. Bernier, D.D.S., M.S., Chief of the Dental and Oral Pathology Section of the Army Institute of Pathology, Maxwell Finland, B.S., M.D., Assistant Professor of Medicine, Harvard Medical School, Louis Fox, D.D.S., South Norwalk, Connecticut, Irving Glickman, B.S., D.M.D., Professor of Oral Pathology and Periodontology, Tufts College Dental School, Milton Greenblatt, A.B., M.D., Director of Laboratory and Research of the Boston Psychopathic Hospital, Irving R. Hardy, D.M.D., Professor of Prosthetic Dentistry, Tufts College Dental School, Robert S. Harris, Ph.D., Director of Nutritional Biochemistry Laboratory, Massachusetts Institute of Technology, Herbert H. Kabnick, D.D.S., New York City, Herbert R. Margolis, D.M.D., Professor of Graduate Orthodontics, Tufts College Dental School, Francis P. McCarthy, M.D., Professor of Oral Medicine, Tufts College Dental School, Clyde Schuyler, D.D.S., F.A.C.D., Diplomate American Board of Prosthodontics, Jerome Schweitzer, B.S., D.D.S., New York City, Harry H. Shapiro, D.M.D., Assistant Professor of Anatomy, College of Physicians and Surgeons, Columbia University, David Weisberger, M.D., D.M.D., Associate Professor of Dental Medicine, Harvard School of Dental Medicine, and Charles H. M. Williams, D.D.S., B.Sc.(Dent.), Assistant Professor of Periodontology, Toronto Dental School.

Focused upon the fundamental and applied aspects of clinical problems in Periodontology and Oral Pathology, each phase of the curriculum will be authoritatively presented so as to assure comprehensive coverage on an advanced level.

Application and reservation form may be obtained by writing "The Berkshire Conference," Tufts College Dental School, 136 Harrison Avenue, Boston 11, Massachusetts. A check for the tuition, or a deposit of $50.00 should accompany the application. Make checks payable to the Berkshire Conference, Tufts Dental School. The conference is located in the heart of the Berkshires at Lenox, Massachusetts, Eastover is a resort hotel which provides the ideal environment for the combination of concentrated study and recreation. The tuition (payable under G.I. Bill) is $125.00 and hotel room and board (entire conference) is $50.00. There will also be a complete program of activities for the ladies and the cost for them is $50.00.

Seminar on Occlusion

Colorado Dental Foundation
Colorado Springs, Colorado

The Colorado Dental Foundation at Colorado Springs, Colorado, held its annual Seminar from October 2nd to October 8th, 1949. The main theme for discussion was Occlusal Disharmony, its causes and consequences, as well as prevention and treatment. The program featured the following men: *Dr. Harry Sicher*, professor of anatomy at Loyola University Dental School, Chicago, Illinois, discussed the anatomy, function and pathology of the temporo-mandibular articulation, and cleared up many of the confused concepts concerning this part of the masticatory organ.

Dr. John R. Thompson, professor of orthodontia, Northwestern University Dental School, Chicago, Illinois, spoke of the importance of harmony between rest position of the mandible and centric occlusion as well as the inter-occlusal clearance, illustrating his presentation with slides of serial tracings of roentgenograms as well as roentgenograms of the temporo-mandibular joint. Several cases of occlusal disharmony were discussed by Dr. Thompson and Dr. Sicher.

Dr. Clyde Schuyler, former professor of prosthetic dentistry, New York University, New York, presented his method of adjusting occlusal disharmony by grinding the teeth. He demonstrated his technique of determining centric occlusion and premature contacts by wax bites, and eliminating interferences. Methods of occlusal reconstruction also were discussed.

Dr. Bertram Douns of Colorado Springs presented his technique of determining the hinge axis of the mandible with the use of face bow with adjustable parts, as well as proper mounting of casts in an adjustable articulator. He discussed the importance of this procedure.

Dr. Balint Orban of Colorado Springs spoke of tissue changes due to occlusal trauma as seen in animal experiments as well as in human autopsy material, discussing the role occlusal trauma plays in periodontal diseases.

Dr. William Hiatt of Denver demonstrated his technique of occlusal grinding.

Other similar Seminar meetings are planned by the Colorado Dental Foundation in the future.

Prior to the Occlusion Seminar, Dr. Orban conducted a microscopic class in histopathology of the supporting tissues of the teeth and a class on theory and practice of periodontal diseases.

University of Illinois College of Dentistry Announces Two-Day Postgraduate Course in Theory and Practice of Periodontics

Dr. Isaac Schour, associate dean in charge of postgraduate studies of the University of Illinois College of Dentistry announces a 2-day postgraduate course in "Theory and Practice of Periodontics" on Friday and Saturday, February 10 and 11, 1950. This course is given for the convenience of the many dentists from out of Chicago who will attend the Midwinter Meeting of the Chicago Dental Society.

Television will play a major role in the presentation of this course. It will be used for the technical and surgical demonstrations. This is perhaps the first time that television has been used as part of a prescribed course in teaching dentistry.

The tuition fee for this course is $25.00. If interested, kindly enroll immediately. The attendance will be limited. Please make your check payable to University of Illinois, College of Dentistry Postgraduate Studies, 808 South Wood Street, Chicago 12, Illinois.

The course comes under the provisions of the G.I. Bill.